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PHILIPPINE DOWNY MILDEW OF MAIZE

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During the past 20 years there have been reported from the Orient several downy mildew diseases of maize, sugar cane, and other economic grasses, caused by members of the genus Sclerospora of the Peronosporaceae. The most recently noted of these has been found in the Philippine Islands, where it causes very serious damage to maize, a crop which in area under cultivation is second only to rice. In 1916, a brief note by Prof. Baker (*1*)[2], of the College of Agriculture, first mentioned the occurrence and destructive power of the disease. In 1918, a short description of it with drawings of the causal fungus was published by Reinking (*17*). No further information concerning this dangerous disease has been published, but it is known that it occasions heavy and constant losses in the maize crop of this the richest of our oriental possessions and represents a grave potential menace to this extremely valuable crop of our own country.

The danger of the introduction of this disease to the cornfields of America was felt to be sufficiently grave to warrant a full investigation in the Philippines and elsewhere in the Orient. By such research it was expected to determine the distribution and life history of this organism and to devise methods of control. With these data in hand, the chances of promptly checking the disease were greatly increased should it gain a foothold in the United States at any time in the future. In the meantime, a quarantine was established against the importation of corn from the Orient.

It was the privilege of the writer to be detailed to this investigation, and since April, 1918, he has been at work on it in the Philippines.

The following paper deals with the general features of the disease and with the characteristics of the causal Sclerospora and its systematic position

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[2] Reference is made by number (italic) to "Literature cited," p. 121-122.

or relationship to other downy mildews destructive to cereal, forage, and sugar-cane crops in the Orient.

DISTRIBUTION

Broadly speaking, the disease is distributed throughout the Philippine Islands. Through the personal observation of the writer and through information given by Dr. Reinking, of the College of Agriculture, by his students, and by members of the Bureau of Agriculture, the disease is known to exist in the Cotobato Valley of the Island of Mindanao at the south, in the Islands of Cebu and Occidental Negros, and in the provinces of Batangas, Laguna, Rizal, Cavite, Bulacan, Tarlac, Pampanga, Nueva Ecija, Pangasinan, Ilocos Norte, la Union, and Isabela in Luzon at the north. In some of these localities the disease appears to have been present for more than 10 years, but as yet not enough is known to warrant a discussion of its probable origin.

DESTRUCTIVENESS

The disease is unusually destructive. It is impossible for one accustomed only to the comparatively light losses occasioned by the maize diseases of the United States corn belt to form any conception of the epidemic intensity of the attacks of this downy mildew under favorable conditions, or of the terrible destruction which it occasions (Pl. 16). Of the aggregate loss to the $8,820,000 maize crop of the Philippines no estimate can be made, because farmers do not recognize the trouble as a disease but regard it as the result of excessive rain or other unfavorable conditions and accept it with fatalistic resignation. In Laguna and Batangas, however, where maize is a major crop and where the writer has studied the disease in the native fields, losses of 40 to 60 per cent are frequent, and in some cases as high as 82 per cent of infection has been counted. In the experimental and acclimatization plots at the College of Agriculture, where the growing of unacclimatized varieties and the constant presence of actively infecting plants combine to make the conditions especially favorable for infection, the losses ordinarily are high. In several beds of United States sweetcorn, planted during the rainy season, every plant was killed before producing any seed.

The severity of the disease in the individual corn plant varies with conditions from the extreme stunting and weakening of the plant resulting in death about one month after planting to the less virulent attacks in spite of which the plant shows a fair growth and ultimately produces a small, more or less poorly formed ear. Even in the few lightly affected cases the grain production is not nearly normal, and in most cases complete barrenness or premature destruction occurs, so the aggregate loss in the average field attacked by the mildew is large. In some localities corn growing has been abandoned for the culture of upland rice because of the ravages of the disease. Moreover, this loss can not be offset in part

by using diseased plants for fodder, for cattle appear to dislike the taste and will not eat the infected plants unless they are mixed with a liberal proportion of the healthy.

One of the most serious features of the attack by the downy mildew is that the infected plants are rendered susceptible to the attacks of a number of secondary parasitic organisms which contribute to the destruction of the weakened plants. In the rainy season there frequently occur destructive rots of the stem, ear, and shank, with which at least two species of Pythium and bacteria appear to be associated, while a species of Helminthosporium, which is only occasionally severe on healthy plants, is usually very destructive to plants weakened by downy mildew.

SYMPTOMS

The effect of the disease on the corn plant varies greatly with such conditions as the age of the plant when infected, the means by which infection takes place, the varietal nature and individual condition of the host, and the environmental conditions which accompany and follow infection. As a result, no small, clearly defined group of symptoms can be described which will entirely cover the effects of the disease on the host.

In general, however, the disease may be said to manifest itself by the loss of chlorophyll in more or less sharply defined areas of the leaf, by the production of a whitish down of conidiophores principally on the chlorotic area, and by a more or less extensive alteration in the form or the normal growth of the plant. The change in color is the most striking and obvious symptom. Since, however, somewhat similar changes in color and form may result from other causes, the characteristic downiness is the surest indication of the disease.

The effects of the disease may appear at any time from the putting out of the third or fourth leaf to the formation and maturing of the tassel and ear, but in any case the tissue of the host is thoroughly invaded by the mycelium before any external signs appear.

When appearing early in the development of the plant the symptoms are as follows: The second, third, or perhaps the fourth leaf, when nearly developed, shows at the base two or three rather narrow, longitudinal stripes of a pale yellow to whitish color (Pl. A) with the exception of which the leaf is quite the normal green. However, the two or three leaves already partly developed above this, and all the leaves which subsequently appear, are almost completely whitish or pale yellow. Moreover, these leaves never attain the normal shape and size but remain much narrower and become rigid, so that they ascend stiffly instead of bending in the normal flexible manner (Pl. A). The growth of the stem is also checked, so that the plant becomes more or less dwarfed. As the growth of the leaf sheaths is not decreased proportionately, they often deeply overlap to form a cover which may inclose and even project beyond the stunted tassel (Pl. 18, A). The root system also is usually affected so that

it does not develop properly but becomes stunted and functionally inadequate.

The subsequent fate of an infected plant varies with conditions. In the rainy season it almost invariably succumbs rapidly to secondary infections by species of Pythium, Helminthosporium, or Fusarium. In the dry season, however, although it usually turns brown, withers, and soon dies, such a plant may struggle along to the tasseling state and may even produce a stunted ear with occasionally a few grains.

When the disease appears later in the development of the corn plant the symptoms are as follows: The first leaf to show any signs of the disease, which may be the fourth or fifth or even up to the eighth, will have at the base pale stripes similar to, but more extensive and broader than, those described for plants which show the disease early (Pl. B). All subsequent leaves show a somewhat similar striping but in a progressively more marked degree, the markings on each successive leaf being more extensive than those on its predecessor and running nearer the tip, while the last leaves are striped throughout their entire length.

The shape of the stripes varies greatly. On the lower leaves they are usually merged at the base into a solid yellowish white area from which irregular elongations run up into the normal green towards the tip of the leaf (Pl. B). On the middle leaves the solid yellowish white area at the base is somewhat smaller in extent, but the prolongations from it run more nearly to the tip, while on the upper leaves these discolored stripes extend from the base to the tip of the leaf but are more broken and irregular and even merge laterally and anastomose so that a marbled or mottled appearance is given to the otherwise green leaf.

The shape and size of these leaves, however, is very little altered, and they usually have the breadth and flexibility which characterize leaves of the normal plant. At times, however, the midribs become brittle from the invasion of the fungus mycelium, break where they join the sheath, and hang straight down along the stem (Pl. 17). The growth and structure of the stem are often normal, and the root system is strong and well developed. It is in the reproductive structures of these later-infested plants that the injurious effect of the disease is shown especially. The tassel, although usually appearing at the normal time and often seemingly unharmed structurally, may show decreased production of pollen and frequently is extremely malformed (Pl. 17, A). The ear also is even more seriously affected. Even a mediocre ear is a very rare occurrence (in 1 out of 150 diseased plants), while customarily the ear is more or less completely sterile and malformed (Pl. 20).

This malformation of the reproductive structures is of frequent and regular occurrence in maize infected by the Philippine downy mildew. In plants attacked at all ages by the disease there is induced a great variety of the most remarkable malformations and monstrosities of the ear and tassel. These show a wide range of the fasciations, phyllodies,

reduplications, virescences, and other abnormalities of the various categories of monstrous growths that are recognized in teratology. Less frequently also the vegetative parts of the infected plants show abnormalities induced by the disease, fasciations and torsions of the stem (Pl. 17, B) and shank (Pl. 19, A) being most common. These abnormalities, of course, are frequently induced by other diseases and by unfavorable conditions of the environment, but their occurrence in connection with the downy mildew is so common as to form an accessory symptom of diagnostic value.

One other marked effect of the disease is the delaying of ear production. Normal plants in a plot invariably will bear well-developed ears in the "milk" or "glazing" stage before the diseased plants have developed ears to the "silking" stage.

It should be noted that the loss of chlorophyll and the consequent yellowish or whitish color of the marked areas, which is so characteristic a symptom of the disease, is by no means permanent but serves particularly to point out the earlier stages of the attack. As the diseased plant matures, however, and the fungus begins to terminate its period of spore production, the marked areas become more and more green, the contrast between the normal green and the paler portions of the leaf becoming less and less distinct until, finally, in plants less heavily attacked, the marked areas may so far regain their green color as to be almost indistinguishable from the normal.

All these plants which show the disease at a late date do not necessarily undergo rapid destruction as in the cases of early attack. On the contrary, although the plants are more susceptible to the secondary infections than are their healthy companions, they may mature along with them, drying and withering at a date only slightly earlier than normal. In some cases the infected plants seem stimulated by the downy mildew to prolonged activity and show persistent and excessive growth of husks, or of bracts in the deformed tassels, after adjacent plants are withered and dry (Pl. 19, A).

The susceptibility to infection is greatest in the young seedling and decreases markedly as the plant develops, so that by the time it has tasselled and is forming ears its tissue is, as a rule, too mature and resistant to permit infection. If, however, as is frequently the case in some varieties, the main plant sends out secondary shoots or suckers, these may rapidly become infected (Pl. 19, B), and through them the infection may spread to the main plant even though it is so far matured as to have its kernels hardening.

When attacked in this way, the mature plant shows symptoms different from any of those described above. The lower leaves are inconspicuously marked throughout their length with narrow, pale, yellow-green to rusty green stripes, which are not continouous but are irregularly broken and interrupted. On the middle leaves, as a rule, the markings

are similar in character but occupy principally the more distal part of the leaves, while the upper leaves are either entirely unmarked or have the striping confined to a small part of the leaf tip. Since most of the parts of the plant are matured, they show no change in form as a result of the infection, but the ear, if not mature, may elongate slightly and project from the husks at the tip (Pl. 19, B). It is to be noted that a plant thus attacked, in contrast to those previously described which are infected early, is marked least extensively and conspicuously on the upper leaves and most extensively on the lower leaves, has ears little if at all altered, and bears no conidiophores on the marked areas.

The production of conidiophores on the diseased plant is, of course, a symptom valuable in recognizing the disease (Pl. 21). Unfortunately, however, the process of conidiophore formation takes place almost exclusively at night and is controlled largely by conditions of the environment. The details of this relationship will be given later. It need only be said here that a plant may be attacked heavily by the fungus, the mycelium of which invades its tissues throughout, and may show the changes of color and growth which are characteristic of the disease and yet never form conidiophores and conidia unless external conditions are favorable.

A comparison of the symptoms of the Philippine downy mildew with those which characterize the downy mildew of maize in other countries shows many similarities.

In the closely related Javan mildew of maize, Palm (*15*) has recognized three distinct sets of symptoms. Of these, the symptoms of type A correspond in general to the description given above for plants attacked early in life, while the symptoms of type B correspond to those of plants attacked later. Type C, however, is characterized by narrow, inconspicuous stripes of a dark brown color running the full length of the lowest leaves and decreasing in extent on successively younger leaves until the last marked leaves show these stripes at the tip only, while the still later leaves are of the normal green throughout.

No specimens corresponding exactly to the description and illustrations of Palm's type C have been seen in the study of the Philippine maize mildew. Occasional stripings of this sort have been observed on the lower leaves of plants whose upper leaves showed the general or restricted discoloration already described. Maturing plants infected through suckers have shown inconspicuous, dark orange-colored stripings, extensive on the upper leaves and decreasing in area on the lower. In no case, however, has an immature plant been seen with these dark markings of the leaves decreasing on successively younger leaves until the latest are untouched.

For the Philippine maize mildew it does not seem justifiable to attempt to make such hard and fast categories as the types A, B, and C of Palm, although the symptoms shown by many plants can be more or less

roughly grouped under the types described above. The discolorations, growth changes, and other effects of the disease all differ markedly in accordance with time of infection, the varietal and individual character of the plant attacked, and the conditions of the environment. Hence there are encountered not only such diseased specimens as can be included conveniently in the three types recognized by Palm but also some plants which show symptoms intermediate between the types and others which show various combinations of these symptoms.

The occurrence of such sharply marked categories of symptoms as those described by Palm might with some justice be suspected to be the manifestation of different biologic strains of the causal fungus. In the Philippine maize-mildew, however, cross inoculations with spores from infected plants corresponding to Palm's symptom types, as well as biometric studies of the spores and conidiophores from these plants, disprove this assumption. Moreover, a series of experiments in which several varieties of maize were inoculated in various ways at different ages and subjected to different environmental conditions, although not entirely completed, has shown that the changes of color and growth produced in the plant by the disease differ with variations in these factors. All the evidence of field observations also supports this conclusion. In general, then, while the symptoms of the Philippine maize-mildew resemble those of the Javan, they appear to be much more varied and less easily grouped into sharply defined categories.

In the related Formosan downy mildew, Miyake (*14*) has described in detail only the symptoms shown by attacked sugar cane, which is the host most severely affected. He states that in maize the stripes are not particularly pronounced and the plant is not noticeably hindered in growth, for it ripens and shows only a slight decrease in yield. While this description would fit occasional plants attacked by the Philippine maize-mildew, it by no means depicts adequately the injurious effects in even the average case and would seem to indicate that the Formosan mildew is far less destructive to maize than is the Philippine.

Upon comparing the maize downy mildew of the Philippines with that of British India described by Butler (*4*), it is to be noted that the symptoms of the latter resemble in general those seen in the Philippines, although in the Indian disease more emphasis is laid on the checking of the internode growth and the consequent stunted appearance of the attacked plants than seems to be warranted from observation in the Philippines. Moreover, although the maize-mildew of British India has been present in that country since 1911, it has, in marked contrast to the Philippine disease, caused only slight sporadic injury.

HOSTS

Under field conditions throughout the Philippine Islands maize is the only crop on which the downy mildew occurs with sufficient severity to attract attention or to occasion appreciable loss. In the trial plots at

the College of Agriculture, however, where many kinds of cereal, forage, and cane crops were grown under conditions favoring infection, the downy mildew was found also to attack teosinte (*Euchlaena luxurians* Schrad.) and sorghum (*Andropogon sorghum* (Linn.) Brot.).

With teosinte, the percentage of infection and resulting loss is not quite so great as with maize, and the symptoms are less pronounced (Pl. 22, C), since the attacked individuals, especially those showing the disease late in their development, are much less conspicuously marked and are very seldom appreciably deformed, while the conidiophores are more scattered and more scantily produced.

As might be expected, hybrids resulting from the crossing of maize and teosinte are also susceptible to the disease, the degree of susceptibility and the effect on the plants attacked being intermediate between those shown by the two ancestors.

In sorghum the percentage of infection is very low, and the few plants infected are easily overlooked, because they turn pale when still very young (Pl. 22, B), bear but few conidiophores, and wither and die after a brief period of weak, stunted growth. No cases of individuals more conspicuously marked or deformed, in which the disease appeared later, were ever seen; and the loss was limited to the destruction of the few attacked plants.

Cross-inoculation experiments and the biometric study of spores and conidiophores show that the same causal fungus is involved in all these cases.

In view of this condition, it would naturally be suspected that other members of the Maydeae and Andropogoneae might also prove susceptible to the disease. So far, however, in spite of extensive search, no such Sclerospora, characterized by a conspicuous and rapidly spreading conidial stage, has been found in this region under natural conditions on the many wild grasses related to maize. However, the writer has found on *Saccharum spontaneum* L., a very common wild grass here, a Sclerospora which although of very frequent and widespread occurrence produces only the characteristic thick-walled resting spores. Further description of this Sclerospora will be given in a later paper, but it should be said at this point that this oogonial form on wild grass does not appear to be connected with the conidial form growing on cultivated maize, sorghum, and teosinte.

Moreover, inoculations such as were successful in the case of maize, sorghum, and teosinte have so far failed to accomplish the transfer of the disease to other related Gramineae—namely, *Coix lachryma-jobi* L., Philippine, United States, and Hawaiian strains; *Coix ma yuen*, Philippine and United States strains; several varieties of sugar cane (*Saccharum officinarum* L.), uba or Japanese cane (*Saccharum* sp.), and the native grasses, cogon (*Imperata cylindracea* L.), anias (*Andropogon sorghum* var. *halepense* L.), and aguingay (*Rottboellia exaltata* L.). In view of the

difficulties in securing artificial infection, these negative results are by no means conclusive, although the successful infection of maize, sorghum, and teosinte under the same conditions would seem to indicate that these other relatives are far less susceptible.

These inoculations will be detailed more fully in a later paper, but it should be said here that they were made from about 2 a. m. until dawn because the spore production was found to take place at this time. It seems highly probable that spore production is nocturnal in the other related downy mildews of the Orient as well and that the uncertain results of inoculations with them has been due to the failure to use fresh spores.

It is of interest to compare these results with those obtained for the other related downy mildews of the Orient. In Formosa, Miyake (*14*) successfully transferred *Sclerospora sacchari* T. Miy. from sugar cane to maize and teosinte and vice versa, but was unable to infect rice, sorghum, wheat, or millet. In India, although their infection experiments were unsuccessful, Butler (*3*) and Kulkarni (*10*) note the occurrence on teosinte of the conidial stage of a Sclerospora which they suspect may be identical with that of maize (*Sclerospora maydis* (Rac.) Butler.)

In Java, no extensive attempts were made by either Rutgers (*19*) or Palm (*15*) to obtain artificial inoculation of other hosts with the Javan downy mildew of maize (*Sclerospora javanica* Palm). They state, however, that under conditions favoring infection in the field, neither sugar cane nor the common wild alang-alang grass (*Imperata* sp.) was found infected and that, although teosinte itself is immune, the hybrid between teosinte and maize is, if anything, more susceptible than the variety of maize from which it is derived.

CAUSAL ORGANISM

The fungus which causes this extremely destructive disease of maize in the Philippines belongs to the Peronosporaceous genus Sclerospora, as Baker (*1*) and Reinking (*17*) already have reported. It should be noted, however, that it shows especially close relationship, not to the type species *Sclerospora graminicola* (Sacc.) Schroet., which is distinguished by the germination of the conidia by zoospores and the abundant production of oospores, but to those other oriental members of the genus which are characterized by the germination of the conidia by tubes and the partial or complete lack of oospores. However, setting aside the question of the affinities of the fungus for a later discussion, its characteristics will now be considered.

MYCELIUM

As a rule, as soon as the maize plant shows any external indication of the disease, the mycelium is found to be quite generally distributed throughout the host tissue, the root being the only main organ which is not extensively invaded. Thisin vasion is most marked in the vegetative

parts of the plant but to a lesser degree affects the male and female inflorescences also.

Since the mycelium is relatively inconspicuous, its course throughout the host tissue is followed with difficulty. However, by means of transverse and longitudinal sections cut in various thicknesses and stained with iron-alum-haematoxylin and eosin or with gentian violet or methyl blue it was possible to trace the relation of the hyphae to the host tissue. Moreover, by subjecting such sections to the processes of maceration, clearing, and subsequent staining used by Mangin (*13*) and Berlese (*2*) the host tissue was readily dissociated and cleared sufficiently to permit the examination of large sections of the mycelium. By these methods material was studied from all parts of plants in various stages of infection, and the nature of the hyphae, their relation to the host tissue, and their location and abundance in different parts of the host were ascertained.

The hyphae are most abundant in the discolored areas of the infected leaves but may be found throughout the plant in unmarked parts of the leaves, in the branch tips of the apparently unaffected tassel, and at the base of the seemingly healthy stem some feet below the first discolored leaf.

In the leaf sheaths, leaves, and such modified foliar structures as the husks and glumes, the mycelium is most abundant among the cells of the bundle sheaths and in the mesophyll tissue (Pl. 23, E), but occasional hyphae are found in the fundamental tissue and even among the elements of the bundles themselves.

In the stem, ear shanks, cob, and tassel rachis, the mycelium follows the bundles, running for the most part parallel to them among the cells of the bundle sheath (Pl. 23, B) and less frequently sending out hyphae more extensively into the surrounding fundamental tissue.

In badly infected ears the mycelium usually runs out from the cob along the funiculus of attachment into the undeveloped parts of the abortive kernels, and occasional hyphae are encountered even in the chaff, seed coats, and endosperm of the apparently healthy kernels, though not in the embryo itself.

Wherever found, the hyphae are almost invariably intercellular in position, occupying even the smallest spaces between the cells, and even forcing adjacent cells apart as they grow between them. Occasionally hyphae were seen which apparently passed within the cells, but the interference of the host tissue was such that their position could not be ascertained with entire certainty. Since the size and shape of the hyphae are determined to a large extent by the nature of the intercellular spaces which they occupy, there is very little regularity in these characteristics in most cases. When separated by maceration, the hyphae are seen to be of two general types—namely, the long, slender, occasionally branching hyphae which lie alongside the vascular bundles in the stem and leaves'

and the lobed, contorted, irregularly branched, gnarled, and crooked hyphae which run in and out among the mesophyll cells of the leaves (Pl. 23, A).

The first kind seem to serve for communication from one part of the host to the other and can be followed for considerable distances even in longitudinal section (Pl. 23, B). The second kind appear to act as a means of establishing connection with the mesophyll cells, especially with the bundle sheath, in order to derive nutriment therefrom, since they are found in every possible crevice in the most intimate contact with the host cells (Pl. 23, A, E).

Haustoria are produced by both types of hyphae but are best developed or most pronounced on the crooked assimilatory hyphae among the mesophyll cells in the leaf. In shape the haustoria are simple, papillate to tubular (Pl. 23, F, G), as a rule, but they may be somewhat lobed (Pl. 23, H). In no case, however, were such markedly digitate haustoria seen as those figured by Rutgers (*19, Pl. 6*) for the Javan Sclerospora. The haustoria penetrate portions of the host cell wall, against which the hyphae are closely appressed, and project into the lumen. Not only the cells of the mesophyll, bundle sheath, and pith are penetrated, but also occasional cells of the epidermis (Pl. 23, E, *c*) and even the xylem (Pl. 23, E, *b*).

In any case, the haustoria accomplish the penetration of the host cell without occasioning its collapse, although the wall often is wrinkled and the turgidity of the cell decreased, apparently through the extraction of its contents by the parasite. The chloroplasts of the parasitized cells are gradually destroyed through the action of the fungus, with the result that the badly infected areas lose their green hue and assume the pale yellow or whitish color symptomatic of the disease. Occasionally the host cell surrounds the haustoria of the parasite with a thick wall (Pl. 23, F), as if in protective response to the injurious stimulus of the fungus, a condition observed also by Butler (*3*) in *Sclerospora graminicola* (Sacc.) Schroet. on Pennisetum.

The hyphae are hyaline, rarely if ever septate, thin-walled, with granular content, and vary greatly in size, 8 μ being perhaps the most common diameter. The haustoria are similar in structure and usually about 8 μ long by 2 μ in diameter.

In the larger air chambers which underlie the stomata, the mycelium develops somewhat irregular clusters of stout branches (Pl. 23, E, *a*), from which, under favorable conditions, the conidiophore initials arise and grow out through the stomata to produce the conidiophores.

CONIDIOPHORES

Conidiophores may be said, in general, to be produced on any part of the plant save the roots. They occur on the main stem, on the leaves, leaf sheaths, and ear husks, and on the main axis, branches, and glumes

of the tassel. Most commonly, however, the conidia appear on the leaves and leaf sheaths, where they occupy principally the conspicuous mottled and discolored areas which have been described.

On whatever part of the plant they may be found, the conidiophores emerge at night, provided there is present a thin layer of dew, rain, or mist. Damp air alone does not seem to permit their formation. Under favorable conditions the process of conidiophore emergence and conidia production begins about midnight and may continue a few hours after dawn, provided the weather is favorably rainy. When seen at night in the luxuriance of their growth, the innumerable conidiophores projecting slightly from the thin film of moisture on the leaves form a very distinct grayish white down, which is by no means even suggested by the dry, matted fragments which remain when the hot morning sun has dried the surface of the leaves (Pl. 21, A).

This process of conidiophore development and conidia production has never been described, and, since it shows several points of interest, it will be presented in detail in a subsequent paper. In general, however, it occurs as follows:

From the stomata of the infected portion one or more club-shaped hyphae grow out. These elongate, and under favorable conditions the paired protrusions finally bud out from their tips and become the stout primary branches. From the tips of these in turn bud out the beginnings of the secondary, and from these, at length, the tertiary branches, each of which usually terminates in one or two tapering sterigmata. Since the initial protrusions which develop into the branches arise almost invariably in pairs, the structure of the mature conidiophore is characteristically dichotomous, instances of the suppression or delayed formation of a branch being, on the whole, rather rare (Pl. 24, D). Finally, from the tip of each sterigma there buds one conidium as a spherical protrusion which enlarges and lengthens until it attains the elongate oval or rounded oblong shape of the mature spore and is separated from the sterigma tip by a cross wall.

When fully formed the conidiophore appears as in Plate 24, C, and consists essentially of a main axis which begins with an elongate basal cell and broadens gradually until it divides into the two to four stout main branches. From each of these extend two to four smaller secondary branches, each of which in turn bears two to four tertiary branches that terminate severally in one or two tapering sterigmata, each bearing at its tip a conidium. Although under favorable conditions the conidiophores are of the large, well-developed type just described, they frequently show such variations in structure as the omission of the second and third series of branches and a general reduction in branches, sterigmata, and consequently number of conidia (Pl. 24, E). On vigorous conidiophores 32 to 96 conidia may be borne, while on poorly developed ones there may be as few as 8 or even 3. In size also there is great varia-

tion, the total length of the conidiophore even in abundant dew varying from 260 to 400 μ, although most commonly it is about 340 μ, while in scanty dew, such as occurs in the hot season, lengths of 160 to 200 μ are generally encountered. In either case, however, the greatest width, just below the branches, is from 15 to 26 μ. The sterigmata are consistently about 10 μ in length, with a diameter at the base of about 6 μ.

The basal cell is invariably present in the mature conidiophore, forming a structural feature which should be emphasized as distinctive (Pl. 24, H, J, L). This cell reaches its greatest width at the septum which separates it from the rest of the main axis and tapers gradually downward throughout its length, terminating in a rounded, slightly swollen foot which is connected by a slender hypha with the internal mycelium through the stomatal pore. The greatest width of the basal cell is usually about 12 μ, but the length varies from the customary extremes in heavy dew (60 to 120 μ) to 30 or even 20 μ in a scanty film of moisture (Pl. 24, E).

When fully mature the conidia are most commonly elongate, ellipsoid, elongate ovoid, or rounded cylindric in shape, are thin-walled and hyaline, and have a more or less finely granular content. The tip is broadly rounded and lacks any papilla or other modification, while the base shows an apiculus, a slight thickening and protrusion of the wall at the point of attachment to the sterigma. Wide variations in the shape of the conidia are common, examples being found of all of the types from subspherical, pyriform, or even lemon-shaped to the extremely elongate types which are shown in Plate 25, C–I.

A method has been devised by Rosenbaum (*18*) for expressing quantitatively the shapes encountered in a study of large numbers of conidia of Phytophthora. This method, which consists in classifying and plotting the ratios of length to width, is of value in that it gives a quantitative idea of the relative predominance of certain shapes of conidia in a species and furnishes a reliable basis for comparison with others. Unfortunately, however, this method can make no distinction between conidia which are ovate or obovate, pyriform, or obpyriform, ellipsoid, allantoid, or cylindrical, provided their length and greatest diameter be the same. Therefore, while the ratios of length to width of 400 conidia of the Philippine Sclerospora of maize are presented here in tabular and diagrammatic form for comparison with other species, a clearer idea of the variations in shape is probably to be obtained from the figures in Plate 25.

The size of the conidia also varies greatly. When large numbers are examined, examples are found with such widely different dimensions as to include those given for several other species. It is difficult, therefore, to give a correct impression of the size of the conidia by means of the extreme dimensions within which they vary, or even by means of the average dimensions. However, the method of grouping together large numbers of representative conidia into a series of measurement classes and plotting curves to show their frequency of occurrence has been used

successfully in describing the size of similarly variable bodies, first by Rosenbaum (*18*) for Phytophthora and more recently by Gaumann (*6*) for Peronospora. This method seems especially valuable in the case of such variable structures as the conidia of the Peronosporaceae, since by means of it data gathered from large numbers of individuals may be so presented that the range of variation in size which is encountered, as well as the size class which predominates in the species, is at once apparent. Also it furnishes a most accurate method for comparing the sizes of such bodies in different species.

TABLE I.—*Measurements and ratios of length to width of 400 conidia of Sclerospora philippinensis arranged in classes.*

Number of conidia in 400.	Length classes.	Number of conidia in 400.	Width classes.	Number of conidia in 400.	Ratio of length to width classes.
	μ.		μ.		
1	17 to 18.9	1	11 to 12.9	1	1.05 to 1.14
1	19 to 20.9			0	1.15 to 1.24
2	21 to 22.9	8	13 to 14.9	3	1.25 to 1.34
1	23 to 24.9			6	1.35 to 1.44
4	25 to 26.9	41	15 to 16.9	9	1.45 to 1.54
10	27 to 28.9			30	1.55 to 1.64
35	29 to 30.9	160	17 to 18.9	53	1.65 to 1.74
68	31 to 32.9			59	1.75 to 1.84
75	33 to 34.9	148	19 to 20.9	70	1.85 to 1.94
64	35 to 36.9			59	1.95 to 2.04
55	37 to 38.9	41	21 to 22.9	40	2.05 to 2.14
30	39 to 40.9			32	2.15 to 2.24
24	41 to 42.9	1	23 to 24.9	21	2.25 to 2.34
21	43 to 44.9			9	2.35 to 2.44
7	45 to 46.9			2	2.45 to 2.54
2	47 to 48.9			3	2.55 to 2.64
0	49 to 50.9			0	2.65 to 2.74
1	51 to 52.9			3	2.75 to 2.84

For these reasons this method seems well adapted to depict the size of the conidia of the Sclerospora of Philippine maize. Accordingly the measurements of 400 conidia are given in tabular form and are also plotted as curves (fig. 1, 2). The ratio of length to diameter in classes is given in figure 3. These show clearly that while spores are encountered with such widely differing dimensions as 18 μ long by 12 μ in diameter, and 51 by 23 μ, the size which predominates is 34 μ by 18 μ, and by far the greater number of spores encountered are from 27 to 39 μ in length by 17 to 21 μ in diameter. Although these measurements are of conidia produced on maize, they have been compared and found to agree with similar measurements of conidia from teosinte and sorghum. On comparing like tabulations of dimensions of fresh conidia with those from material mounted in glycerin or dried, the writer finds constant slight differences, particularly in width. Therefore, these 400 measurements were made on four occasions at the beginning of the period of maximum conidia production (2 to 3 a. m.) from fresh material mounted in dew

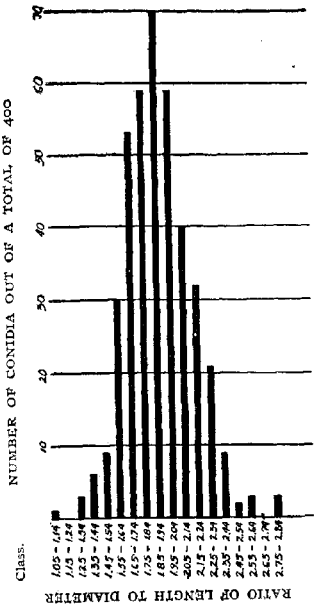


FIG. 3.—Diagram showing ratios of length to width of conidia of *Sclerospora philippinensis*, arranged in classes, and indicating limits of variation and mode.

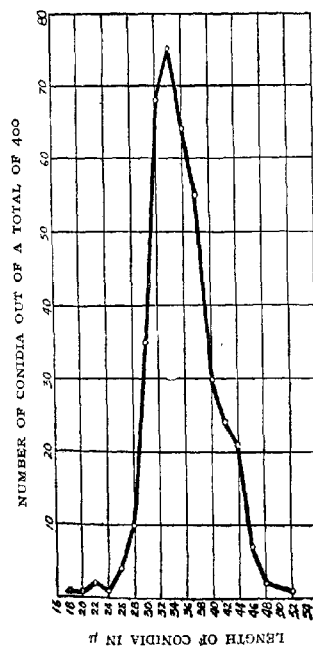


FIG. 1.—Graph showing variation in length of conidia of *Sclerospora philippinensis*.

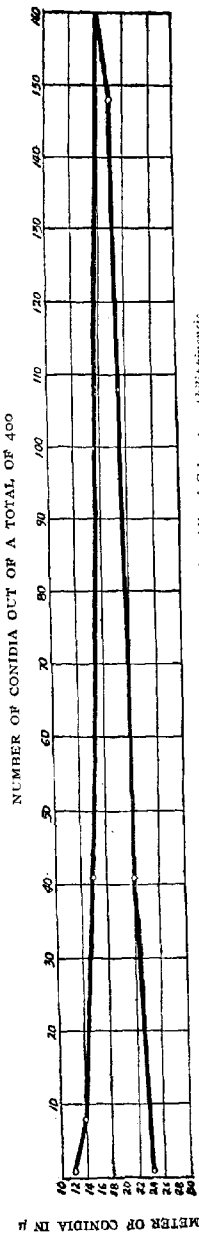


FIG. 2.—Graph showing variation in diameter of conidia of *Sclerospora philippinensis*.

or rain water, and they probably furnish an expression of the conidia dimensions and a means of comparison with other species which is as accurate as it is possible to obtain.

It is interesting to note that occasional monstrous conidia were seen, resembling somewhat those described by Miyake (*14*) for *Sclerospora sacchari*. Since these have the same structure and history as those of more usual size and appear to represent merely the upper extreme of the widely varying conidia dimensions, they are regarded by the writer as of no special significance.

Germination of the fresh conidia takes place readily in dew, in rain water, in water from clear brooks, in dilute nutrient solutions of various kinds, and on similar solutions solidified with 1 per cent agar. When once the conidia are dried, however, they will no longer germinate under any conditions. On the moist surfaces of newly infected plants in the field, large numbers of conidia may be found germinating vigorously at any time from about 3 a. m. until dawn, but after the rapidly drying effect of the early sun has been felt for one or two hours there can be found on the same plants only shrivelled spores incapable of further development. Germination is preceded by a swelling and consequent alteration in size and shape of the conidium and invariably proceeds by the protrusion of one or more germ tubes (Pl. 25, C–L). This may take place from any part of the spore, and the hyphae thus produced may simply elongate (Pl. 25, C, J) or develop variously into extensively branching systems (Pl. 25, E, F, L). Occasionally the hyphae of germination grow up into the air for a short distance and produce at their tips an ovoid swelling that might perhaps be interpreted as an abortive attempt at a secondary conidium formation such as has been found in other Peronosporaceae. In no case was the production of zoospores by the conidia observed, although repeated attempts were made to induce this method of germination.

In spite of the ease with which the conidia produce germ tubes, all attempts to induce continued independent development of the mycelium in artificial media have been unsuccessful, the growth seemingly ceasing when the nutriment of the spore is exhausted. In view of the tropical habitat of this Sclerospora it is of interest to note that the conidia germinate readily when maintained at a temperature as low as 6.5° C., even though the temperature at which they most commonly germinate is from 20 to 24°.

In spite of extensive search, none of the resting or resistant bodies customarily encountered in this or other genera of the Peronosporaceae, such as chlamydospores and parthenogenetic or normal oogonia, have ever been found to be associated with the conidial stage of this fungus. Every effort has been made to find such structures. The progress of the disease has been observed in individual plants from the time of their infection by the fungus to their ultimate disintegration in many varie-

ties representing the several types of maize and teosinte and in sorghums under all the various conditions of the wet, dry, and transitional periods of the year. Furthermore, infected plants have been subjected to various changes in temperature, moisture, light, aeration, and soil, in the attempt to induce the formation of such structures. So far, all efforts have been in vain, and, although facilities were not available for any such experiment as subjecting the infected plants to long-continued cold or total freezing such as might occur in our own corn belt, still the experiments which were made seem to indicate that the formation of resting bodies by the fungus in maize occurs very rarely if at all under the conditions naturally encountered in the Philippine Islands.

The possibility that the conidial stage may be restricted to maize while the production of oogonia takes place on some other host invites consideration. As has been mentioned above, the writer has found a Sclerospora attacking a common field grass, *Saccharum spontaneum*, in this region; but whether this fungus, of which only the oogonial stage has been seen, is in any way connected with the conidia-bearing Sclerospora on maize remains to be determined.

IDENTITY OF THE CAUSAL FUNGUS

The important question of the identity of this Philippine Sclerospora necessitates a comparison with the other members of the genus. Since our knowledge of the Philippine form is at present confined to its conidial phase solely, no comparison is possible between it and those species of which only the oogonial stage has been recorded, such as the remarkable *Sclerospora magnusiana* Sor. (*20*) of Equisetum from Russia, the rare *Sclerospora farlowii* Griff. (*7*) of Chloris from western North America, the recently described *Sclerospora miscanthi* T. Miy. (*14*) of Miscanthus from Japan, or even the more common *Sclerospora macrospora* Sacc. (*8*), which is widely distributed on a large number of grasses and even has been found on the tassels of maize in Italy. Likewise, the type species *Sclerospora graminicola* (Sacc.) Schroet., although known from all over the world on a wide range of wild and cultivated grasses and even recorded on maize in Argentina (*21*), can not be directly compared, because the conidial stage, even though known, is rare and is characterized by the germination of the conidia by zoospores and by the invariable predominance of the typical oospores.

A far closer relationship is shown between the Philippine form and those Oriental species which occur on maize or related gramineous hosts and are characterized by the partial or complete lack of an oogonial stage, with the concomitant predominance of the conidial phase, which is distinguished further by the germination of the conidia by tubes.

Of these there have been enumerated the following: *Sclerospora javanica* (Rac.) Palm, of Java (originally described by Raciborski as *Peronospora maydis*); *Sclerospora maydis* (Rac.) But., of India; and

Sclerospora sacchari T. Miy., described by Miyake from Formosa but reported by Lyon (*11*, *12*) also in the Fiji Isalnds and Queensland.

It has been assumed by Baker (*1*) and Reinking (*17*) that the Philippine Sclerospora of maize is identical with *Sclerospora maydis* (Rac.) But. of India, and this has been generally accepted by other investigators. Since no detailed description of the species with critical measurements has been published, and the single conidiophore and few spores figured by Reinking are hardly enough on which to base a decision, it seems necessary to corroborate the identification of the fungus.

A comparison with *Sclerospora maydis* (Rac.) But. of India and also with the other related species mentioned above is accordingly in order. Such a comparison must necessarily consider the field characters of the disease, such as its effect on the plants attacked, its severity, and its fatality to the various hosts, as well as the specific peculiarities of the causal organism itself. Of these the characteristic structure and dimensions of the organism itself are most valuable, since the field characters show, on the one hand, a general similarity in all these fungi and, on the other hand, vary so widely under different conditions as to be confusing even in one species.

The Sclerospora causing the Philippine disease is known in its conidial phases only, and a comparison of this form with other species must be based on this stage. Such a comparison is confronted by many difficulties. In the first place, the characters most valuable from the systematic point of view have been found by the writer to vary greatly under different conditions and at different stages in the development of the Philippine form, and they probably do so in the other forms also. For instance, the very important characters of the size and shape of the conidia and the structure and dimensions of the conidiophores vary greatly at different stages of development and under different conditions.

The conidia begin as small spherical outgrowths from the sterigmata tips, and in their development become larger and more elongate, passing through ellipsoid (Pl. 24, A), oval (Pl. 25, A), and even pyriform stages before they eventually assume the elongate ovoid ellipsoid or rounded cylindrical shape of complete maturity (Pl. 24, C). They are then separated from the sterigma tip by the septum.

This characteristic shape is transient, however, for, after they are free from the conidiophores, the spores show an almost immediate imbibition of moisture, which results in a marked increase in size and in a more rotund shape, due to the greater bulging of the side walls. Moreover, the apiculus which marks the point at which the spore was attached to the sterigma is modified, by the swelling of the spore, to a low, rounded curve.

Since the partially developed spores of various shapes and sizes may be detached from the sterigmata and still retain their contents and germinability, and since marked changes from the shape and size of the

mature spores normally follow when it is free, it is obvious that a mount of spores usually comprises a motley collection of shapes and sizes, only a comparatively small number of which represent the characteristics of the normal and mature spore.

Moreover, aside from these variations which mark the normal development of the spore, there are also changes in size and shape resulting from abnormal conditions such as the sudden checking of development by unusual drying of the necessary layer of moisture on the leaf.

The size and structural characteristics of the conidiophores also vary markedly with attendant environmental conditions. The normal order is for primary, secondary, and tertiary branches to form before the sterigmata develop and begin to bud out the spores. If the gradual drying of the film of moisture on the leaf surface begins to check this process before its completion, however, sterigmata formation and spore production ensue prematurely, and conidia may be borne on the secondary or primary branches of the conidiophore (Pl. 24, I), or even on the apex of the main axis itself. Similarly, the growth of the basal cell and main axis may be curtailed (Pl. 24, E). Obviously, as a result of these changes, the height of the conidiophore shows a corresponding alteration.

Finally, after it has lost its conidia, the conidiophore shrivels and is dried to an almost unrecognizable mummy by the morning sun.

Since it appears highly probable that similar variations in size and structure occur also in the other oriental mildews, it is difficult to make any adequate comparison from the data available. To permit accurate comparison one should have descriptions and illustrations of material, or the material itself, collected under the optimum conditions, which in the case of the Philippine downy mildew occur on cool nights with heavy dew or persistent rain from 2 to 4 a. m.

In the light of this fact, Miyake's (*14*) data are valuable, as he recognized that conidiophores and conidia were produced at night, and his drawings show that he illustrated excellent material. Most investigators, however, failed to realize this, and their material, as their descriptions and drawings show, was inadequate and scanty.

When one compares the available data, inadequate though they be, the following points are apparent. The Philippine and Javan Sclerosporas are alike in that the conidial phase is the only one yet known.

The conidiophores of the former closely resemble those of *Sclerospora javanica* Palm both in size and structural characteristics, such as the basal cell, the main axis, the branch system, and the ultimate sterigmata. On the contrary, the conidia of the two forms are noticeably different. In the Javan fungus they are oblong rotund in shape and measure 19 to 26 μ in length by 15 to 20 μ in diameter, while in the Philippine mildew they are elongate ellipsoid, elongate oval, or rounded cylindric and markedly longer, most of the conidia encountered measuring about 34 μ in length

by 17 μ in diameter, and comparatively few showing the shortness which marks the Javan form. Moreover, although the field characters of the two diseases are very similar, the Javan Sclerospora presents an additional point of difference in that it does not attack teosinte, although teosinte-maize hybrids are, if anything, even more susceptible to it than maize itself (*19*).

To *Sclerospora sacchari* T. Miy., of Formosa, the Philippine maize mildew shows a very close resemblance in the size, the form, and even the minor structural characteristics of the conidiophores. Also, the conidia of the two forms are evidently quite similar, since the illustrations and the description (ellipsoid or oblong with rounded apex, 25 to 41 μ long by 15 to 23 μ in diameter) of the Formosan conidia are applicable to those of the Philippine species also. A marked difference between the two, however, is shown in their virulence on various hosts, for, while *Sclerospora sacchari* grows on both maize and teosinte as does the Philippine Sclerospora, still the former attacks sugar cane of many varieties, including those grown most commonly in the Philippine Islands, with violent intensity, while the latter, so far as is known, does not infect that crop at all. In the Philippines, in regions heavily infected with the maize-mildew, sugar-cane fields comprising many varieties grown under widely varying conditions and situated adjacent to the badly infected maize, and even containing some maize plants growing among and in contact with the young cane, have been under frequent observation during all stages of their development for over a year, and yet no case of infection with the downy mildew of maize has ever been seen.

Moreover, inoculation experiments such as were successful with maize, teosinte, and sorghum have so far failed to cause infection of the Philippine Sclerospora of maize on sugar-cane varieties found susceptible to the Formosan disease. Furthermore, the oogonial stage which has been reported for *Sclerospora sacchari* T. Miy. forms an additional point wherein it differs from the Philippine fungus, although it should be noted that the oogonia, which have been found only once and are not figured, have not been proved to be connected with the conidial stage of *Sclerospora sacchari*.

On comparing the Philippine downy mildew of maize with the British Indian species (*Sclerospora maydis* (Rac.) But.), with which it has been regarded as identical, a close resemblance indeed is apparent. The conidia especially are similar in both shape and size in so far as one can judge from the data available; the lack of any other type of spore is another point of agreement. In considering the conidiophores of the former, however, it should be noted that the description, dimensions, and illustrations indicate that the material was imperfect, for if one may judge from the Philippine fungus, the size and the abruptly ending base of the conidiophore signify that the main axis had been broken off just above the basal cell. Any accurate comparison, therefore, is difficult. The

sterigmata, however, are comparable, and it is clear that those of the British Indian fungus are markedly larger (15 to 20 μ long) than those of the Philippine species.

Moreover, the field characteristics are noticeably different. Although Butler reported the first attack of the disease at Pusa in 1912 and emphasized the probability of its spreading to other fields of the region, his latest report (5) indicates that it has continued to be only slightly and restrictedly destructive, an effect markedly in contrast to the rapid spread and serious damage of the Philippine fungus. Also, Butler's description emphasizes the stunting of the growth and resultant bunchy appearance of the plant as a characteristic feature of the disease in India, while in the Philippines this is but one and certainly not the most striking effect of the disease.

While the matter is necessarily unsettled because of lack of adequate description of the British Indian form, certain points would seem to indicate that the maize-mildew of India is a different physiological variety and probably a different species from that of the Philippines. These points are the differences in the causal fungi and the symptoms, and especially the lack of virulence shown by the Indian disease and its failure to spread through Bengal where "maize is a crop of considerable importance" and where the conditions of climate and culture are little if at all different from those of some infested regions of the Philippines.

In any case, however, it should be noted that the name *Sclerospora maydis* (Rac.) But. is not strictly a tenable one, for it was applied to the British Indian maize-mildew by Butler (*3, p. 15*) on the assumption that it was identical with the Javan. Butler (*4, p. 275–276*) concluded from his comparison with the diagrammatic drawings and incomplete descriptions of Raciborski (*16*) that the downy mildew of maize in British India—

> was found to be identical with the one which causes great damage to this crop in Java,

and that—

> its cause is a fungus named *Peronospora maydis* by Raciborski.

The more recent and extensive work of Palm (*15*), however, has shown clearly that the Javan fungus, although indeed a Sclerospora, is a distinct species, one which Palm names *Sclerospora javanica*. This leaves *Sclerospora maydis* (Rac.) But. as the name of the British Indian maize-mildew.

Therefore, because the points of difference already considered seem to indicate that the downy mildew of maize in the Philippines is not identical with the one in British India, and because the name *Sclerospora maydis* (Rac.) But., given to the latter, is technically untenable, it seems necessary to distinguish the Philippine downy mildew of maize. Hence it is

given the name of *Sclerospora philippinensis*, n. sp., with the diagnosis as follows:

Sclerospora philippinensis, n. sp.[1]

Sclerospora Maydis, Reinking, 1918, *in* Philippine Jour. Sci., s. A, v. 13, no. 5, fig. 39, pl. 20, fig. 1–2, not Butler.

Forming linear or irregular whitish yellow to pale spots, often entirely discoloring the leaves and more or less deforming the host.

Mycelial hyphae growing intercellularly in all parts except the root, branched, slender, usually about 8 μ in diameter, but irregularly constricted and inflated, haustoria simple, vesiculiform to subdigitate, small, about 8 μ long and 2 μ in diameter.

Conidiophores always produced in night dew and growing out of the stomata, erect, 150 to 400 μ long, 15 to 26 μ thick, bearing a basal cell in the lower part, dichotomously branched two to four times above, branches robust, sterigmata conoid to subulate, 10 μ long, slightly curved.

Conidia elongate ellipsoid, elongate ovoid, or rounded cylindrical, varying in size, usually 27 to 39 μ long by 17 to 21 μ broad, hyaline, with thin episporium, minutely granular within, slightly rounded at the apex, provided with a minute apiculus at the base, always germinating by a tube.

Oospores not yet seen.

Material of the type has been deposited in the pathologic collections of the Bureau of Plant Industry, Washington, D. C., in the Cryptogamic Herbarium at Harvard University, Cambridge, Mass., and in the herbarium of the Bureau of Science, Manila, P. I.

So far as at present known there exist in the Orient the following Sclerosporas which are of primary importance, since they cause serious diseases of maize.

Sclerospora javanica Palm, known on maize and maize-teosinte hybrids in Java, Madoerah, and Sumatra.

Sclerospora maydis (Rac.) But., known on maize and teosinte in Bengal, British India.

Sclerospora sacchari T. Miy., known on maize, sugar cane, and teosinte in Formosa, and on sugar cane in Queensland and the Fiji Islands.

Sclerospora philippinensis, n. sp., known on maize, teosinte, and sorghum in the Philippine Islands.

All these species are very similar in their effects and show close relationship in structure and development. All are characterized by the

[1] *Sclerospora philippinensis*, sp. nov.

Maculas lineares vel irregulares, albido-flavas vel pallidas effiiciens, saepe totum folium discolorans, et matricem plus minusve deformans.

Hyphis mycelicis inter cellulas in totas partes praeter radicem crescentibus, ramosis, tenuibus, plerumque circa 8 μ in diametrum, sed irregulariter constrictis inflatisque, cum haustoriis simplicibus, vesiculiformibus subdigitatisve, minutis, circa 8 μ longis et 2 μ in diametrum ornatis.

Conidiophoriis semper in rore nocturno productis, e stomatibus egredientibus, erectis, 150–400 μ longis, 15–26 μ crassis, in parte inferiore cellulas basilares gerentibus, superne 2–4 dichotomo-ramosis, ramis robustis cum sterigmatibus conoideo-subulatis, 10 μ longis, leviter curvatis.

Conidiis elongato-ellipsoideis, elongato-ovoideis vel rotundato-cylindraceis, variis dimensione, plerumque 27–39 μ longis et 17–21 μ latis, hyalinis, episporio tenue, intus minute granulosis, apice leviter rotundatis; basi cum apiculo minute mamitis, semper per tubum germinantibus.

Oosporis nondum visis.

Hab. in foliis, vaginis, glumis, bracteis, culmis, et inflorescentiis praecipue *Zeae maydis*, rarius *Euchlaenae luxuriantis* et *Andropogonis sorghi* per omnes partes in insulis Philippinis.

predominance of the conidial stage, no oospores having been found connected with any save *Sclerospora sacchari*, with which, indeed, the relationship is not very well established.

Furthermore in all these species the conidiophores are large and prominent with a differentiated basal cell, stout main axis, and extensive dichotomous system of branches comprising large primary, secondary, tertiary, and even quaternary branches. The germination of the conidia also is invariably by means of hyphae.

In contrast to these species the cosmopolitan *Sclerospora graminicola* (Sacc.) Schroet., the type on which the genus was established, is characterized by the predominance of the oogonial stage, the conidial phase being comparatively rare; by its smaller inconspicuous conidiophores, which lack a differentiated basal cell and give rise to few short primary or at times secondary branches only; and by the regular germination of the "conidia" by zoospores.

Such marked and essential differences certainly appear to indicate that these oriental species should be separated from the type as a different genus; but, in the opinion of the writer, such a step can not be made with justice until more is known of the conidial stage of *Sclerospora graminicola* and of the oogonial stage of the oriental forms.

Moreover, whether *Sclerospora graminicola* var. *andropogonis-sorghi* Kulk. should be included with the oriental group by virtue of its well-developed conidiophores and the germination of the conidia by hyphae, as Ito (9) suggests, also depends on further knowledge of the points just mentioned.

When one considers the great variations in effect on the host and even in such essential features as the characteristics of the conidiophores and conidia, which have been found by the writer to occur in *Sclerospora philippinensis* under different conditions of the environment at different stages of its development and on various hosts, one can not avoid a suspicion that these oriental forms may in reality be a single species. It is not inconceivable that such may be the case and that the variation in effect on the host, the susceptibility of different plants in different places, and the variations in structure of the causal organism may all be due to environmental conditions of the regions in which they are found. Obviously to settle these important points conclusively there is need of extensive cross-inoculation experiments and of comparative studies, using optimum material and methods which emphasize important characters quantitatively as well as qualitatively.

The problems of the origin of these destructive Sclerosporas of maize and of their geographic distribution, their appearance in the Orient where maize has only been introduced since about 1496, and their absence as yet from the Western Hemisphere where maize originated, are all too involved for consideration at present.

In any case, however, the increasing attention which these dangerous downy mildews of maize have demanded by their destructive activity throughout the Orient in recent years must necessarily arouse the apprehension of all who are concerned with the valuable corn and sugar-cane interests of the United States.

SUMMARY

(1) For several years there has been known to occur in the Philippine Islands a destructive downy mildew of maize, which not only causes serious losses in that region but also threatens our own valuable corn crop, should it reach the United States. Prior to investigations by the writer no extensive study of this disease has been made. This paper presents certain results in regard to the distribution, severity, and characteristics of the disease and the nature and relationships of the causal fungus.

(2) The disease occurs throughout the Philippine Islands, where it evidently has been established for some years.

(3) It is extremely destructive. Under favorable conditions whole fields are destroyed, and in some districts it has even forced the natives to abandon corn culture entirely.

(4) Representative varieties of all types of maize are highly susceptible, and teosinte, maize-teosinte hybrids, and sorghum are attacked, but with less virulence. Inoculation experiments on a number of related plants, both wild and cultivated, gave negative results.

(5) Symptoms of the disease may appear from the time the plants are seedlings with three or four leaves to the time the tassels and silk are developed. In general, infected plants show a yellowing of the leaves in more or less restricted striped areas, a whitish down of conidiophores, principally on the leaves, abnormalities in growth of the vegetative parts, and abortive development of the ear, resulting in partial or complete sterility. These effects of the disease are described and illustrated.

(6) The causal fungus belongs to the genus Sclerospora of the Peronosporaceae and is characterized by the predominance of its conidial stage, the lack of oospores, so far as known, and the invariable germination of its conidia by hyphae. In these respects it differs from the type species *Sclerospora graminicola* (Sacc.) Schroet., which is distinguished by its evanescent conidial stage, its predominating oospores, and the germination of its "conidia" by zoospores. The Philippine species shows close relationship to the following recently described oriental species, all of which attack maize: *Sclerospora javanica* Palm, of Java, *Sclerospora maydis* (Rac.) But., of British India, and *Sclerospora sacchari* T. Miy., of Formosa, Queensland, and the Fiji Islands.

The Philippine Sclerospora appears to be a new species and is described as *Sclerospora philippinensis*, n. sp.

(7) Maize plants usually are infected as very young seedlings, and less often as they mature. In any case, however, when the symptoms appear, the mycelium of the fungus already has invaded the host tissue extensively. The mycelium may be found in practically every part of the maize plant with the exception of the root, but is most abundant among the bundle sheath cells of the leaf.

(8) The conidiophores are produced in vast numbers but only at night when a thin layer of dew or rain is on the leaf surface. They vary greatly in size and development according to the depth and persistence of this layer. These variations are described and figured.

(9) Since the conidia also show wide variation in size and shape, an attempt is made to give a quantitative idea of this by tables and graphs of the measurements of 400 specimens. When fresh, the conidia germinate readily in water and various culture media at temperatures ranging from 6.5° to 25° C., and invariably by hyphae. Once they have become dried the conidia no longer germinate; hence their distribution and the infection of new plants occurs almost always before dawn.

(10) In spite of extensive search, no oospores or other resting bodies have yet been found to be produced by this Sclerospora. It apparently maintains itself by transmission from plant to plant. The writer has found the oospore stage of a new Sclerospora on *Saccharum spontaneum* L., a common wild grass of the Philippines. Whether this is in any way connected with the conidial stage on maize remains to be determined.

LITERATURE CITED

(1) BAKER, C. F.

1916. ADDITIONAL NOTES ON PHILIPPINE PLANT DISEASES. *In* Philippine Agr. and Forester, v. 5, no. 3, p. 73–78.

(2) BERLESE, E. J.

1898–1904. SAGGIO DI UNA MONOGRAFIA DELLE PERONOSPORACEE (1898–1903). *In* Riv. Patol. Veg., s. 2, v. 6, p. 79–110, pl. 7–10; v. 7, p. 19–37, 1898; v. 9, p. 1–126, illus., 1901; v. 10, p. 185–298, illus., 1904. Reprinted.

(3) BUTLER, E. J.

1907. SOME DISEASES OF CEREALS CAUSED BY SCLEROSPORA GRAMINICOLA. *In* Mem. Dept. Agr. India Bot. Ser., v. 2, no. 1, 24 p., 5 pl.

(4) ——

1913. THE DOWNY MILDEW OF MAIZE (SCLEROSPORA MAYDIS (RAC.) BUTL.). *In* Mem. Dept. Agr. India Bot. Ser., v. 5, no. 5, p. 275–280, pl. 8–9 (1 col.).

(5) ——

1918. FUNGI AND DISEASES IN PLANTS . . . 547 p., illus., pl. Calcutta. Bibliography, p. 518–531.

(6) GÄUMANN, Ernst.

1918. ÜBER DIS SPEZIALISATION DER PERONOSPORA AUF EINIGEN SCROPHULARIACEEN. *In* Ann. Mycol., v. 16, no. ½, p. 189–199, 6 fig. Zitierte Literatur, p. 199.

(7) GRIFFITHS, D.

1907. CONCERNING SOME WEST AMERICAN FUNGI. *In* Bul. Torrey Bot. Club, v. 34, no. 4, p. 207–211. (Continued article.)

(8) IPPOLITO, G. D', and TRAVERSO, G. B.
1903. LA SCLEROSPORA MACROSPORA SACC. PARASSITA DELLE INFIORESCENZE DI ZEA MAYS LIN. *In* Staz. Sper. Agrar. Ital., v. 36, p. 975–996, pl. 9–11.

(9) ITO, Seiya.
1913. KLEINE NOTIZEN UEBER PARASITISCHE PILZE JAPANS. *In* Bot. Mag. Tokyo, v. 27, no. 323, p. 217–223.

(10) KULKARNI, G. S.
1913. OBSERVATIONS ON THE DOWNY MILDEW (SCLEROSPORA GRAMINICOLA (SACC.) SCHROET.) OF BAJRI AND JOWAR. *In* Mem. Dept. Agr. India Bot. Ser., v. 5, no. 5, p. 268–273, pl. 6–7 (1 col.).

(11) LYON, H. L.
1911. NOTES ON THE SUGAR INDUSTRY OF FIJI. *In* Hawaiian Planters' Rec., v. 4, no. 6, p. 318–339, 6 fig.

(12) ———
1915. THE AUSTRALIAN LEAF STRIPE DISEASE OF SUGAR CANE. *In* Hawaiian Planters' Rec., v. 12, no. 4, p. 257–265, 2 fig.

(13) MANGIN, L.
1890. SUR LA STRUCTURE DES PÉRONOSPORÉES. *In* Compt. Rend. Acad. Sci. [Paris], t. 111, no. 24, p. 923–926.

(14) MIYAKE, Tsutomu.
1911. ON A FUNGUS DISEASE OF SUGAR CANE CAUSED BY A NEW PARASITIC FUNGUS, SCLEROSPORA SACCHARI T. MIY. *In* Rpt. Sugar Exp. Sta. Govt. Formosa, Div. Path. Bul. 1, 61 p., 9 pl. In Japanese.

(15) PALM, Bj.
1918. ONDERZOEKINGEN OVER DE OMO LIJER VAN DE MAIS. (With an English summary.) *In* Meded. Lab. Plantenziekten [Batavia], no. 32, 78 p., 7 pl.

(16) RACIBORSKI, M.
1897. LIJER, EINE GEFÄHRLICHE MAISKRANKHEIT. *In* Ber. Deut. Bot. Gesell., Jahrg. 15, Heft 8, p. 475–478, 4 fig.

(17) REINKING, O. A.
1918. PHILIPPINE ECONOMIC-PLANT DISEASES. *In* Philippine Jour. Sci., s. A., v. 13, no. 4, p. 165–216, fig. 1–20; no. 5, p. 217–274, fig. 21–42, 22 pl.

(18) ROSENBAUM, J.
1917. STUDIES OF THE GENUS PHYTOPHTHORA. *In* Jour. Agr. Research, v. 8, no. 7, p. 233–276, 13 fig., pl. 71–77. Literature cited, p. 273–276.

(19) RUTGERS, A. A. L.
1916. DE LIJER-ZIEKTE DER MAIS. *In* Meded. Lab. Plantenziekten, no. 22, 30 p., 7 pl. (3 col.).
Cover-title: De Peronospora-Ziekte der Mais.

(20) SOROKINE, N.
1889. MATERIAUX POUR LA FLORE CRYPTOGAMIQUE DE L'ASIE CENTRALE. *In* Rev. Mycol., ann. 11, no. 43, p. 136–152, pl. 90.

(21) SPEGAZZINI, C.
1909. MYCETES ARGENTINENSIS. SER. IV. *In* An. Mus. Nac. Buenos Aires, s. 3, t. 12, p. 257–458. (Continued article.)

PLATE A[1]

Young maize plant, showing the effects of a very early attack of the downy mildew on a small, early maturing variety, Manobo Yellow. Notice the dwarfing and the pale appearance of the plant as a whole, the narrowness and stiffness of the leaves, and the narrow striping of the later leaves throughout their length. The plant was 32 days old when photographed and had first shown the disease two weeks after emerging from the soil. One-fourth natural size.

[1] In the preparation of Plates A and B the diseased plants were photographed and enlargements were colored to correspond as closely as possible to the living specimens. Prepared by L. S. Weston.

PLATE B

Young maize plants, showing the effects of later attack of the downy mildew on a large, late-maturing variety, Guam White Dent.

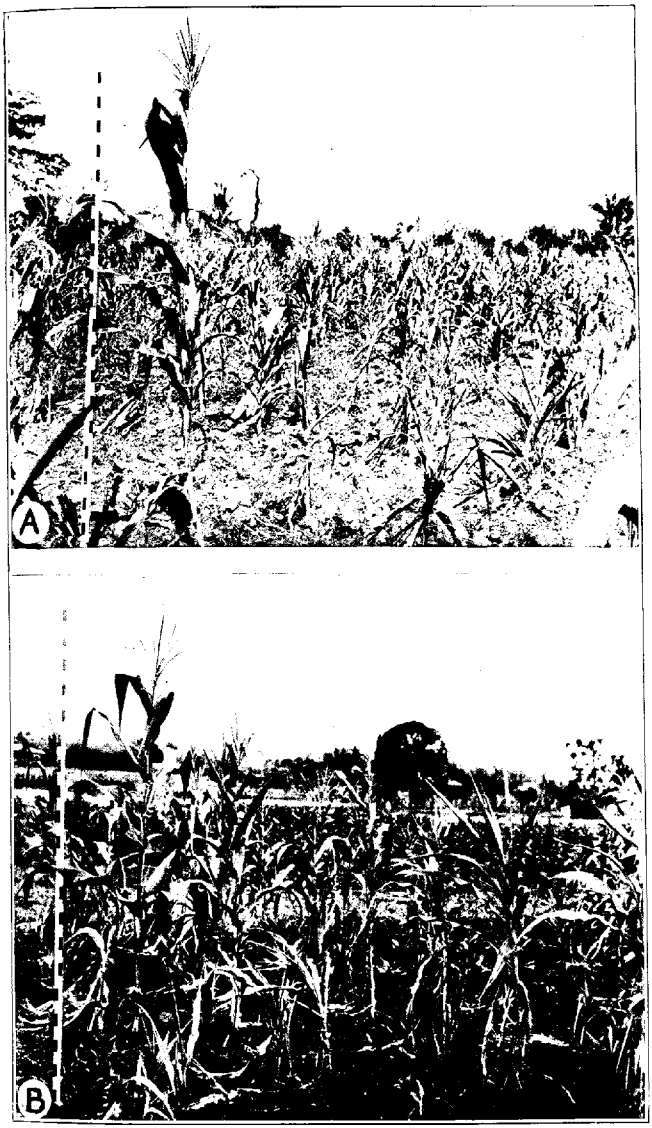
The two plants at the right are diseased; the one at the left is healthy. Notice the characteristic markings on the larger diseased plant—the whitish yellow sheath of the lowest affected leaf, the short narrow stripes at the base of the next leaf, and on the later leaves the whitening of the entire breadth at the base and the extension of broad stripes increasingly far into the normal green of each successive leaf tip. The leaves are nearly as broad and flexible as in normal plants, and their growth is little checked, if at all. The plants are 31 days old and developed the symptoms of the disease 25 days after emerging from the soil. One-seventh natural size.

PLATE 16[1]

A.—Portion of a field of Moro White maize, showing heavy loss from the downy mildew. At the left, near the scale, is seen the only healthy plant that remains in this part of the field. Near it are several pale, stunted plants which are already withering, while in the background may be seen other plants less seriously attacked.

B.—View near the edge of a field of Guam White Dent maize, showing the ravages of the downy mildew. The tall, dark-leaved plant near the scale and two others a little farther back are the only healthy plants seen. Notice the stunted, withering specimens in the foreground, and at the right the seriously affected individual with stiffly ascending, striped leaves.

[1] On the scale which appears in this and the following photographs each black division equals 5 cm. Photographs by W. H. Weston.


A
B


B
A

PLATE 17

A.—A frequently encountered type of downy mildew effect. The maize plant is sterile, with no ear borne in the normal place but with a couple of small, abortive ears growing at the base of the tassel. The leaf sheaths are whitish yellow, and conspicuous stripes of the same color occupy a large proportion of the leaves. These are stiff and brittle, the young ones at the top of the plant ascending at an unnatural angle and the older ones breaking and hanging down stiffly.

B.—A serious case of downy mildew injury, one of hundreds in a badly attacked maize field. The conspicuous striping of the leaves and the crooked stalk at once attract attention. The small ear is sterile. At the base of the plant can be seen another one badly dwarfed by the disease.

PLATE 18

A.—A common result of downy mildew attack. In both maize plants shown the growth of the internodes has been checked so that the leaf sheaths overlap and the unexpanded tassel is still partly surrounded by them. The striping of the leaves and their stiff, brittle character are easily seen. Both plants were entirely barren.

B.—A maize plant seriously injured by the downy mildew stands in front. Its stunted habit and striped leaves are striking evidences of the disease. Of the two abnormal ears, the one at the right was entirely sterile while the one from which the husks have been removed bore a few viable seeds. In the same hill, behind, is a healthy plant, only the lower part of which is shown.

C.—One hill in a maize plot which lost heavily from attacks by the downy mildew. The diseased plant at the left, although nearly as tall as the healthy companion at the right, is less strong and has a poorly developed ear which is only partly inclosed in husks and bears very few kernels.

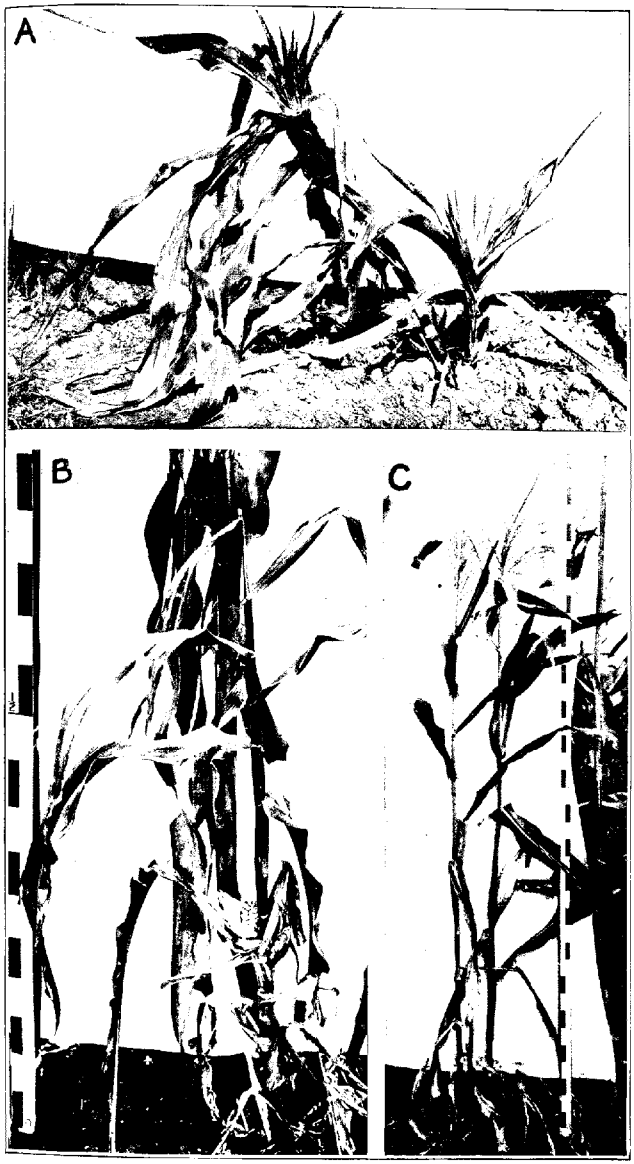

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PLATE 19

A.—A case of abnormal growth of a maize plant as the result of an attack by downy mildew. The shank has elongated enormously, and an excessive development of the husks has taken place. Only a small, completely sterile ear has formed.

B.—A maize plant which, when nearly mature, became infected by the downy mildew through a small sucker previously developed. The sucker is obviously infected, but the large plant, aside from faint leaf stripings which escape the camera, shows the effect of the disease only in its unexpanded tassel and protruding ear tip.

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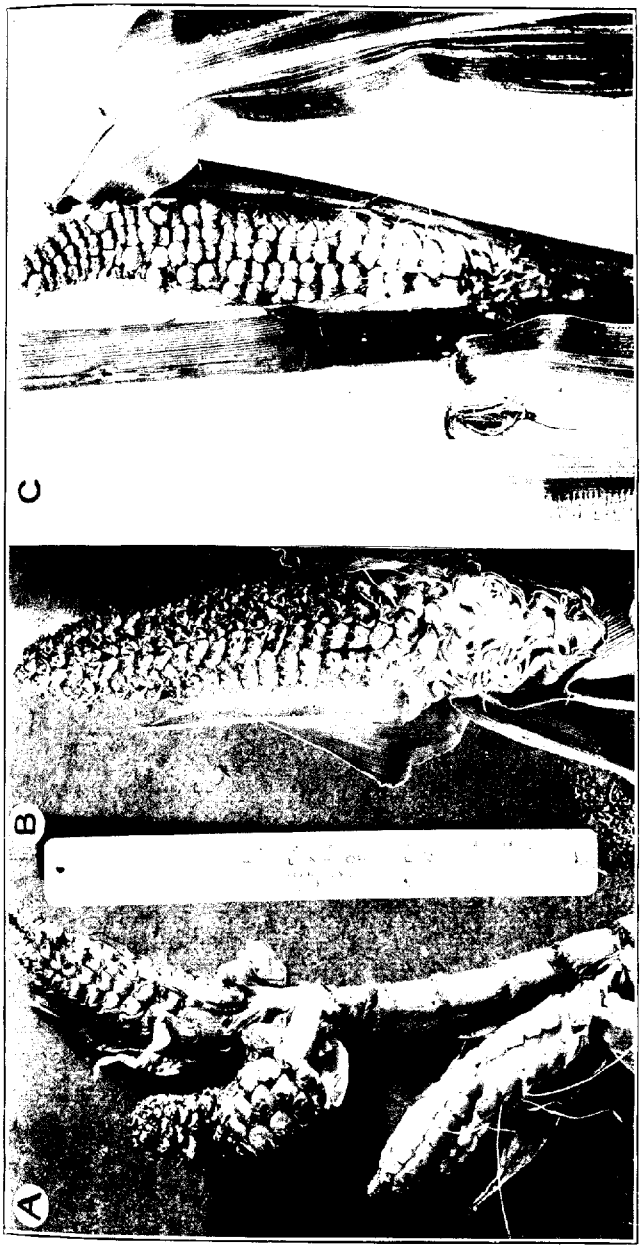
PLATE 20

A.—A deformed and partly sterile ear complex produced by a maize plant as a result of downy mildew infection. Notice the branching and elongation of the shank, the abnormal development and arrangement of many of the kernels, and the inclosing of a few kernels in tunicate bracts. The husks have been removed. This specimen is from a Yellow Dent variety that normally has one large and well-developed ear.

B.—A maize ear developed abnormally as a result of the downy mildew. The husks, beyond which the upper third of the ear protruded, have been partly removed. Save for two or three at the base with partly developed kernels, all the florets were sterile, green in color, and bract-like in texture. Healthy plants of this Yellow Dent variety bear large ears well covered over by husks.

C.—Ear of a maize plant infected by the downy mildew. Only a few viable seeds have been formed, the remainder of the florets being poorly developed and sterile. Notice the conspicuous stripes on the leaves. Before the husks were removed the tip of the ear protruded beyond them. Normally this White Flint variety bears long, well-filled ears.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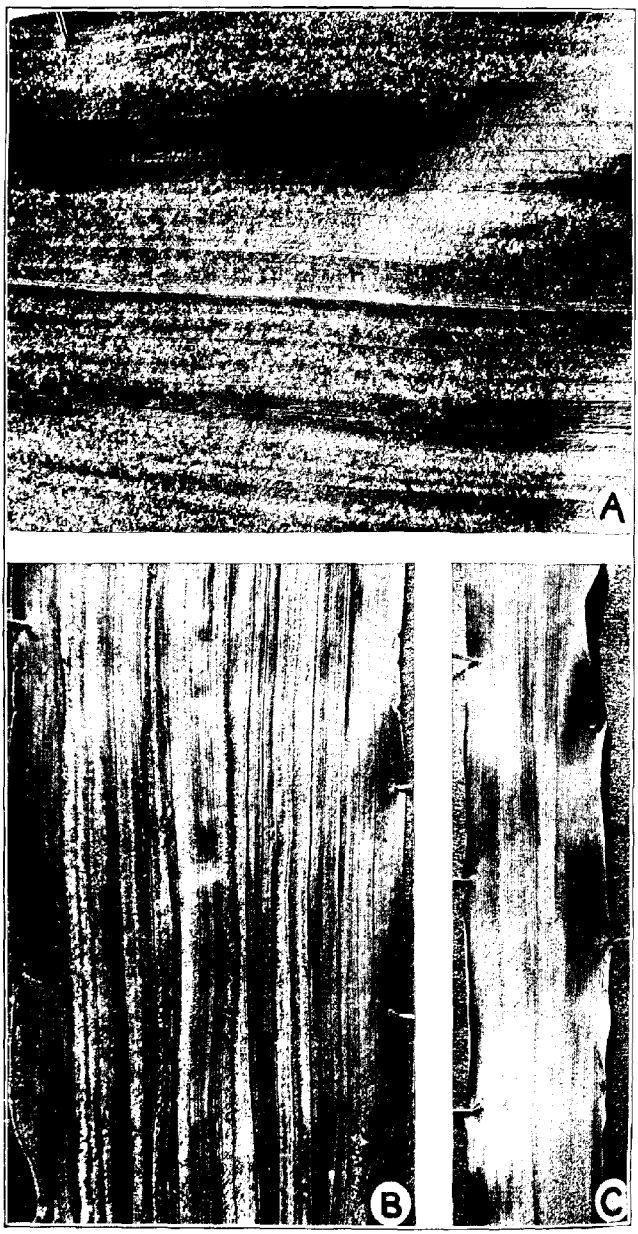
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PLATE 21

A.—A near view of the thick down of conidiophores which has been produced on the upper surface of a badly diseased maize leaf. This gives an idea of the vast numbers of conidiophores which are borne on even a small area. The regions where they are formed most abundantly correspond in general to the stripings of the leaf. The layer of dew in which the conidiophores were produced has just dried. × 2.

B.—Upper surface of a badly infected maize leaf from a maturing plant. Conidiophore production is in this case restricted to the yellowish white stripes like those shown in the colored plate. × 1½.

C.—Upper surface of the middle portion of a maize leaf from a very young plant which has only recently developed the markings of the disease. The stripes are seen to be covered with conidiophores even up to the ends. × 1½.

PLATE 22

A.—View of a row of Egyptian sorghum showing tall, green, healthy plants at the left and at the right a dwarfed, yellowish white plant which is infected by the downy mildew.

B.—Near view of this diseased sorghum plant. Notice the slender, stunted habit, the pallor and faint stripings of the leaves.

C.—A comparative view of healthy teosinte (right), and teosinte seriously infected with the downy mildew (left). The healthy plant has many suckers and is large and vigorous with broad, flexible, dark green leaves and well-developed inflorescences. The diseased plant has no suckers, is stunted and weak, and bears slender, stiff, brittle leaves, which are pale in color and inconspicuously striped, and poorly developed tassels containing a few abortive seeds at the base.


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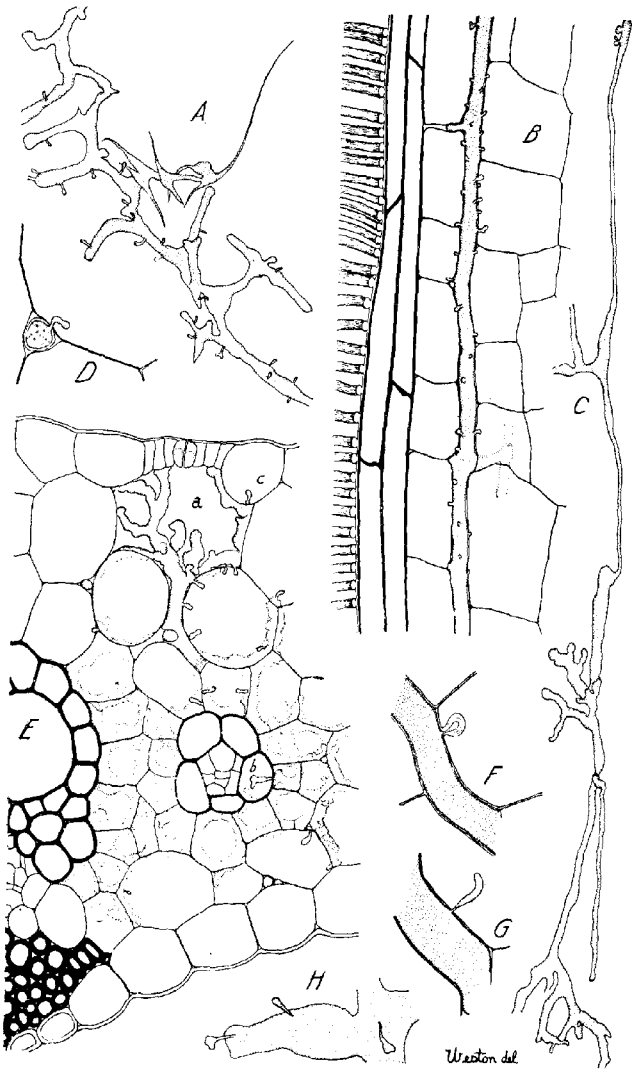
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PLATE 23[1]

A.—Portion of the typical crooked, irregular mycelum with numerous haustoria which is found in the mesophyll of badly infected leaves, here freed from the host tissue by maceration. × 375.

B.—Longitudinal section cut from the center of a maize stem 8 inches from the ground. The plant, over 5 feet in height, was just putting out its tassel and had recently shown markings of the disease on its four uppermost leaves. A strand of the mycelium can be seen running alongside the bundle between cells of the bundle sheath which are penetrated by numerous haustoria. × 375.

C.—Portion of the mycelium freed by maceration from tissue of the midrib at the base of a badly infected leaf. × 375.

D.—Hypha cut in cross section as it lies between three adjacent mesophyll cells of the host. The penetration of a characteristic haustorium into one of the host cells is shown. × 850.

E.—Transverse section from a badly infected portion of a maize leaf, showing the abundant mycelium running between the cells of the bundle sheath and forming in the substomatal air chamber the branches (*a*) that grow out through the stoma to form the conidiophores. The haustoria are seen penetrating not only the mesophyll cells but also a cell of the xylem (*b*) and the epidermis (*c*). × 375.

F.—Portion of a hypha lying between adjacent mesophyll cells, one of which has formed a many-layered wall around the haustorium invading it. × 850.

G.—Portion of a hypha similar to that shown in F but with the haustorium unhindered in its invasion of the host cell. × 850.

H.—Bit of mycelium such as is shown in A but more highly magnified to show the haustoria. × 850.

[1] The drawings were made with the aid of a camera lucida and are all from preserved material of maize.

PLATE 24[1]

A.—Slender, sparingly branched conidiophore bearing comparatively few conidia. It is only partially matured, as can be seen from the small size and rotund shape of the conidia and from the incomplete development of the septum. From maize during heavy dew. × 375.

B.—Tip of branch with two conidia *in situ*. Treated with osmic acid and stained, thus differentiating the two sterigmata as more hyaline than the branch tip. × 750.

C.—Stout, much-branched, mature conidiophore bearing 38 spores. From maize during heavy dew. × 375.

D.—Upper portion of a nearly mature conidiophore with one secondary branch which has failed to branch further and has terminated in a single conidium only. × 375.

E.—Small, stunted, sparingly branched conidiophore produced on maize during the light dew of the hot, dry season. Note the poorly formed cell and the small size and restricted development of the conidiophore as a whole in comparison with those formed in heavy dew, as shown in A and C. × 375.

F.—Basal cell with two thick crosswalls; From maize. × 375.

G.—An unusual basal cell with two septa and an abnormally large footlike base. × 375.

H, J, L.—Typical basal cells of conidiophores. × 375.

I.—Upper portion of an underdeveloped conidiophore bearing three spores on sterigmata arising directly from the top of the main axis. × 375.

K.—Tip of an ultimate branch with two sterigmata bearing conidia. The right conidium is shown as if in optical section, the left in surface view. × 850.

M.—Basal cell of a conidiophore from teosinte with septum formation progressing by the centripetal extension of a cellulose-pectose ring. The footlike projection at the base is abnormally large. × 375.

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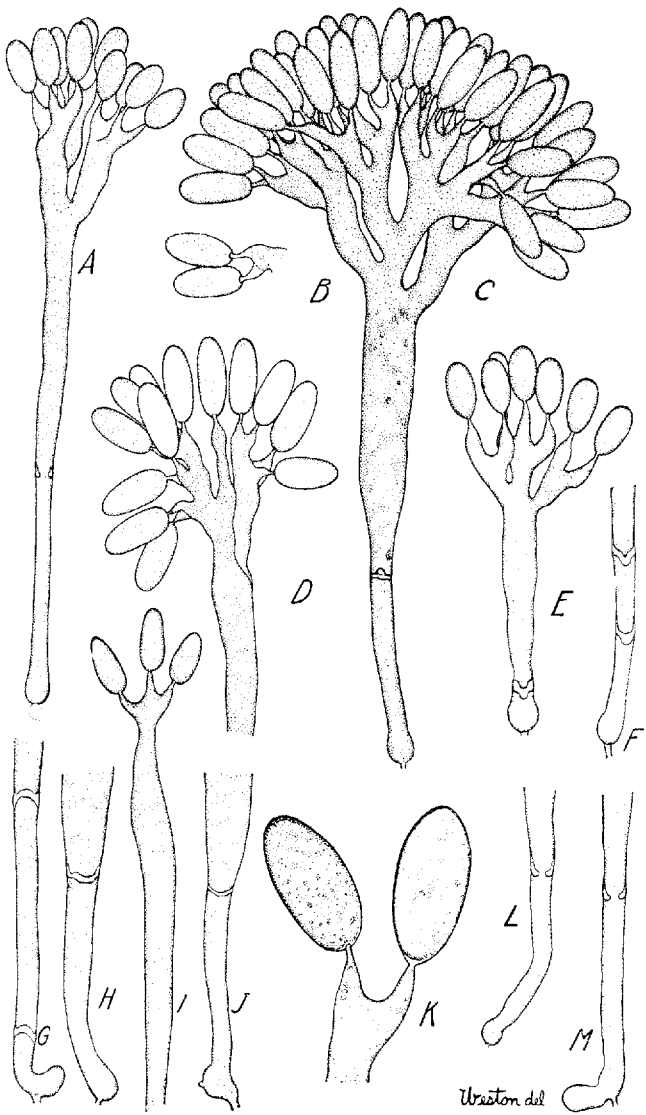
[1] The drawings were made with the aid of a camera lucida and are from fresh material, with the exception of B, G, K, and M. G, J, and M are from material on teosinte; all other figures are from material on maize.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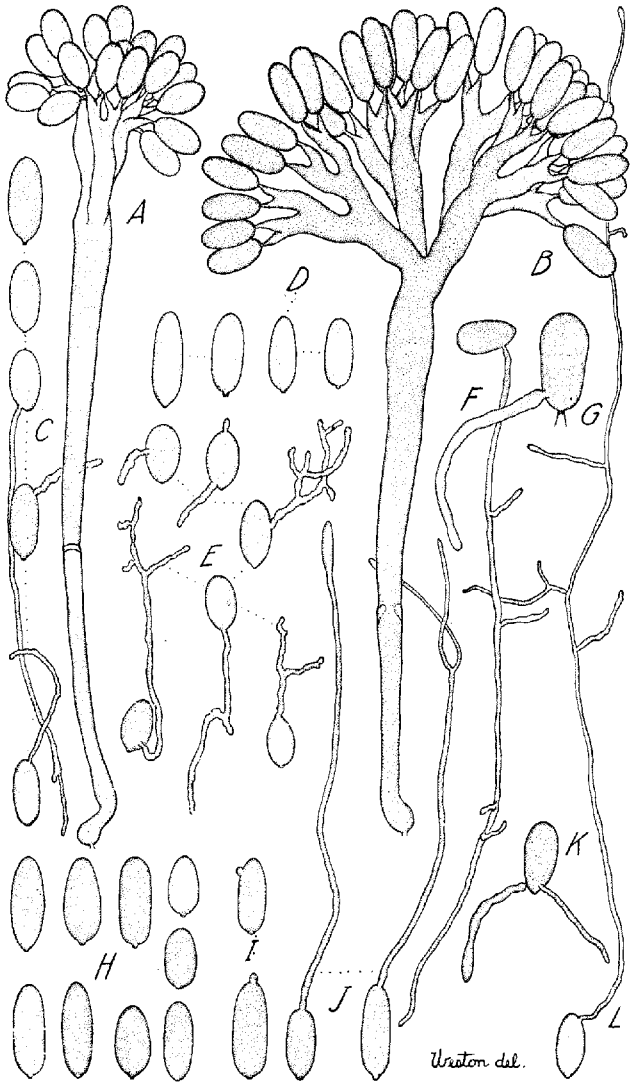
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PLATE 25[1]

A.—Conidiophore from sorghum, partly matured and bearing few conidia. Compare with Plate 24, A. × 375.

B.—Conidiophore from teosinte, nearly mature, with extensive system of branches bearing many conidia. Compare with Plate 24, C. × 375.

C.—Typical conidia from sorghum. Three are germinating in dew by means of relatively simple hyphae. × 375.

D.—Typical conidia from teosinte. × 375.

E.—Typical conidia from teosinte which have germinated in dew on the leaf surface. × 375.

F.—Conidium from teosinte germinating by an extensive branched hypha when maintained in dew at 7° C. × 375.

G.—Conidium from teosinte germinating while still attached to its sterigma. × 500.

H.—Typical conidia from maize, showing common variations in shape and size. × 375.

I.—Two conidia from maize just beginning to germinate in rain water. × 375.

J.—Two conidia from maize germinating in sterilized brook water maintained at 8° C. × 375.

K.—Conidium from maize germinating in dew on the leaf surface. × 375.

L.—Conidium from maize giving rise to extensive branching hyphae in a dilute decoction of young maize kernels. × 375.

[1] The drawings were made with the aid of a camera lucida and are all from fresh material with the exception of A and G.

EFFECT OF DRUGS ON MILK AND FAT PRODUCTION

By FRANK A. HAYS, *Associate*, and MERTON C. THOMAS,
Assistant in Animal Husbandry, Delaware Agricultural Experiment Station

The opinion that milk production and butter-fat yield can be influenced by the use of drugs is widespread among dairymen. Many have their own opinions on this question, and some prominent feeders have been accused of "drugging" test cows. Three of our advanced registry associations now prohibit the use of all drugs during the official test period.

Marshall[1] states that some drugs and feeds are said to increase the milk and butter-fat yield. Friedberger and Fröhner[2] inform us that a number of galactogogues have always been recommended together with a liberal supply of feed largely fluid in character. They mention preparations of antimony, sulphur, chlorate of potash, fennel, juniper berries, caraway seed, aniseed, dill, and common salt. These writers recommend the "milk powder" used as drug No. 5 in the experiment reported below.

The value of an experimental test of different drugs lies not in the fact that it might make possible some abnormal test records in the hands of the unscrupulous but in the fact that it will furnish some information on the relation of feed components to the complex physiological processes of milk secretion.

PREVIOUS WORK

Henderson[3] reports the effects of using six different drugs as galactogoges. Each drug was used on 10 cows, and the period of treatment was either two days or one week, with a control period of equal length either before or after treatment.

Henderson summarizes his results as follows:

1. With sodium bi-carbonate the cows increased the milk yield but neither the fat production nor the per cent. of fat in the milk.

2. With ginger the cows increased the per cent. of fat in the milk but decreased the milk yield and total fat production.

3. With pilocarpine hydrochlor injected into the cows hypodermically in most cases the cows increased both the per cent. of fat in the milk and total milk production.

4. With malt extract the cows in most cases appeared to increase the milk and butter fat production, but it had no effect upon the per cent. of fat in the milk.

5. Neither gentian nor powdered nux vomica had any effect either on the milk production or on the quality of the milk.

6. When grain alcohol was applied to the udder just previous to milking, no effect on the milk production or per cent. of fat in the milk was noted.

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[1] MARSHALL, Francis H. A. THE PHYSIOLOGY OF REPRODUCTION ... p. 566. London, 1910.

[2] FRIEDBERGER, Franz, and FRÖHNER, Eugen. VETERINARY PATHOLOGY. Translated by M. H. Hayes, ed. 6, v. 1, p. 396-397. London, Chicago, 1908.

[3] HENDERSON, Harry Oram. A STUDY OF FORCED FEEDING AND METHODS USED IN ADVANCED REGISTRY FEEDING. *In* Penn. Agr. Exp. Sta. Ann. Rpt. 1915/16, p. 393-419. 1918.

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Key No. Del.-3

McCandlish[1] reports two series of trials with galactogoges. In the first series one cow was used and in the second there were three. The experimental period covered two days in each series, with a control period of two to four days following.

Results as given by McCandlish may be summarized as follows:

1. On the whole, alcohol depressed rather than stimulated milk and butter fat production.

2. Castor oil decreased the percentage of fat in milk, but the changes in milk yield were not appreciable.

3. Pitutarin treatment resulted in decreased milk and butter-fat yield.

4. Administration of pilocarpine and physostigmine resulted in an increased fat yield in the first series. One of the cows in the second series showed an increased fat yield, while the other two showed a decrease in milk yield.

5. The effect of aloes was greatly reduced milk yield and a fat yield somewhat reduced, but the averages show little change.

6. A mixture of epsom salts, common salt, and nux vomica showed only slight effect on milk and fat yield.

THE EXPERIMENT[2]

The experiment was begun April 14, 1919, and closed July 11, 1919. The objects of the experiments were:

1. To determine the effect of various drugs on the butter-fat test of milking cows.

2. To study the effect on the total fat yield of producing cows.

3. To determine whether drugs have an effect on the health or on total milk production.

METHOD

Four cows of mature age were chosen as experimental animals. No. 1 was a grade Holstein, No. 2 was a pure-bred Holstein, and No. 3 and 4 were pure-bred Guernseys. The interval of experimentation with each drug was five days. A control period of five days preceded all experimental periods, except the first five days of the experiment. Each of the four cows received a different drug for a 5-day period. This was followed by a 5-day control period during which no drugs were given. At the end of this period the drugs were shifted so that each cow received a different drug from the one previously given. Thus the control and experimental periods alternated, and the order in which the drugs were given was so arranged that each cow received each of the eight drugs for a 5-day period.

The cows experimented upon were milked twice daily, the weight of milk was recorded, and composite samples of the milk from each cow were tested for butter fat daily.

Drug mixture No. 1 was recommended to us by a prominent dairyman. The mixture No. 5 is one recommended by Friedberger and

[1] McCANDLISH, Andrew C. THE POSSIBILITY OF INCREASING MILK AND BUTTERFAT PRODUCTION BY THE ADMINISTRATION OF DRUGS. *In* Jour. Dairy Sci., v. 1, no. 6, p. 475–486. 1918.

[2] Credit is due Dr. C. C. Palmer for administering drug No. 6 hypodermically.

Fröhner.[1] All the drugs except No. 6 were given mixed with the grain feed twice daily.

DRUGS USED

1. Food tonic consisting of 100 pounds oil meal, 5 pounds saltpeter, 5 pounds epsom salts, 5 pounds gentian, 5 pounds fenugrek, 8 pounds powdered charcoal, and 5 pounds sulphur, fed at the rate of 2 ounces daily per cow in two feeds.

2. Air-slaked lime, fed at the rate of 2 ounces daily per cow in two feeds.

3. Fowler's solution of arsenic, fed at the rate of 2 fluid ounces daily per cow in two feeds.

4. Gentian fed at the rate of 2 ounces daily per cow in two feeds.

5. Tonic mixture consisting of the following: 3 ounces black sulphid of antimony; 1½ ounces sulphur; 5 ounces each of fennel, caraway, and juniper berries, 1 pound common salt, fed at the rate of 2 ounces daily per cow in two feeds.

6. One gr. physotigmine sulphate injected hypodermically daily per cow, ½ grain in two doses.

7. Sodium bicarbonate, fed at the rate of 2 ounces daily per cow in two feeds.

8. Ginger, fed at the rate of 2 ounces daily per cow in two feeds.

EXPERIMENTAL RESULTS

Figures 1 to 8 present graphically the individual milk and butter-fat yield of each cow. A solid line is used for the control period and a dotted line for the experimental period.

Figure 1 shows the results of the tonic mixture. There was a slight increase in fat for the pure-bred Holstein and for one of the Guernseys, but the other cows showed no perceptible change. The milk yield was slightly increased for one Guernsey and slightly decreased for the other three cows.

Figure 2 shows that air-slaked lime increased the fat yield in two cases and the milk yield in two cases.

Figure 3 shows that when Fowler's solution of arsenic was used, two cows increased in fat production and three in milk production.

Figure 4 indicates that powdered gentian has a tendency to increase fat yield slightly but has little effect on milk production.

Figure 5 shows that the German tonic mixture did not increase either fat or milk production.

Figure 6 seems to indicate that physostigmine sulphate has a depressing effect on both milk and fat yield.

Figure 7 unfortunately shows the fat record for only three cows. There is no indication of any appreciable effect of sodium bicarbonate on production

[1] FRIEDBERGER, Franz, and FRÖHNER, Eugen. OP. CIT.

Figure 8 indicates that cows fed ginger begin to decline in production about the second or third day.

Table I gives a summary of results, showing the average of the four cows in total milk and total butter fat and the average test as obtained by dividing the fat yield by the milk yield given in the table.

TABLE I.—*Effect of drugs on milk yield, fat test, and fat yield during 5-day period*

Drugs used.	Average total milk.			Fat test.			Average total butter fat.		
	Control.	Treated.	Gain or loss.	Control.	Treated.	Gain or loss.	Control.	Treated.	Gain or loss.
	Pounds.	*Pounds.*	*Pounds.*	*Per ct.*	*Per ct.*	*Per ct.*	*Pounds.*	*Pounds.*	*Pounds.*
Food tonic No. 1	92.2	93.7	+ 1.5	4.37	4.57	+0.20	3.998	4.285	+0.287
Air-slaked lime	67.3	81.9	+14.6	5.21	5.00	− .21	3.509	4.095	+ .586
Fowler's solution of arsenic	107.3	109.0	+ 1.7	3.72	3.86	+ .14	3.995	4.210	+ .215
Gentian	113.5	108.2	− 5.3	3.93	3.98	+ .05	4.459	4.302	− .157
German tonic mixture	108.8	108.0	− .8	3.97	3.75	− .22	4.205	4.049	− .156
Physostigmine sulphate	108.7	99.9	− 8.8	3.89	3.60	− .29	4.226	3.706	− .520
Sodium bicarbonate	93.1	90.8	+ 2.3	4.43	4.43	+ .00	4.132	4.020	− .112
Ginger	92.5	93.8	+ 1.3	4.28	4.32	+ .04	3.568	4.055	+ .087

Drugs 1, 2, 3, and 8 slightly increased the milk yield, but this increase is insignificant except when air-slaked lime was fed. The increase of 21.7 per cent for the air-slaked lime group we think is significant. Gentian and physostigmine sulphate seem to depress the milk yield, and the German tonic mixture No. 5, and sodium bicarbonate have but little effect.

The fat test was appreciably increased by tonic No. 1, by lime, and by Fowler's solution. There was significant decline in fat test shown by the groups fed the German tonic mixture and physostigmine sulphate.

Average total butter-fat production was probably significantly increased by air-slaked lime. Food tonic No. 1 and Fowler's solution show increase of 0.28 and 0.21 pound, respectively, in fat for the 5-day period. A decline of 0.52 pound is shown by the physostigmine sulphate group. The decline in other groups is not considered significant.

No difficulty was encountered in getting the cows to take any of the drugs, and no effect on their physical condition was observed.

SUMMARY

(1) A study of individual records and average records does not indicate that drugs have a very pronounced effect on the production of the dairy cow.

(2) Air-slaked lime fed in 2-ounce doses daily may possibly increase milk production and total fat yield.

(3) No other drug or mixture tested proved to be of value to increase production.

(4) Results do not indicate that the difference in character of milk of Holstein and Guernsey cows has any relation to their manner of reaction to drugs.

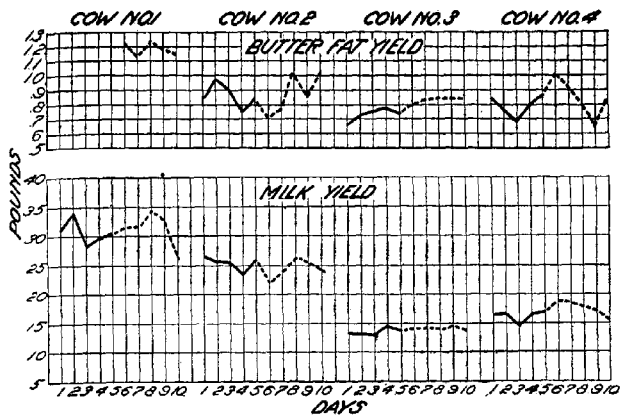

FIG. 1.—Graph showing effect of tonic mixture No. 1 on butter-fat and milk yield.

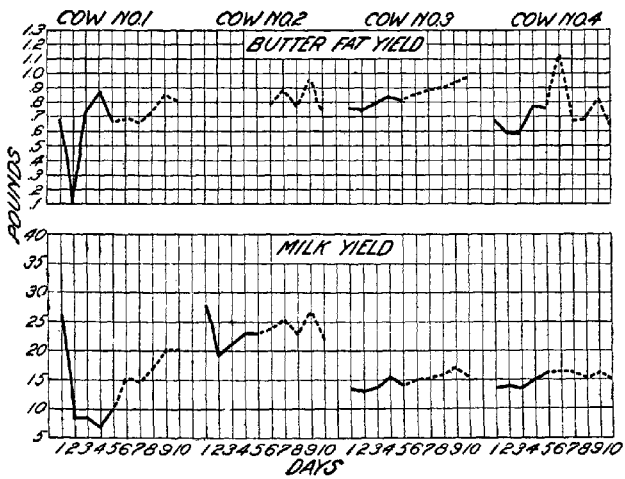

FIG. 2.—Graph showing effect of air-slaked lime on butter-fat and milk yield.

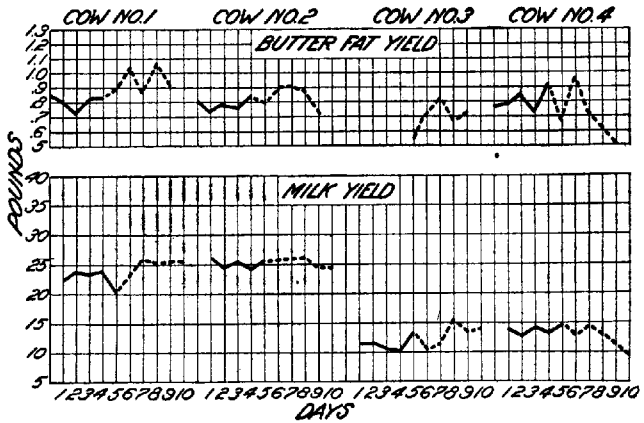

FIG. 3.—Graph showing effect of Fowler's solution of arsenic on butter-fat and milk yield.

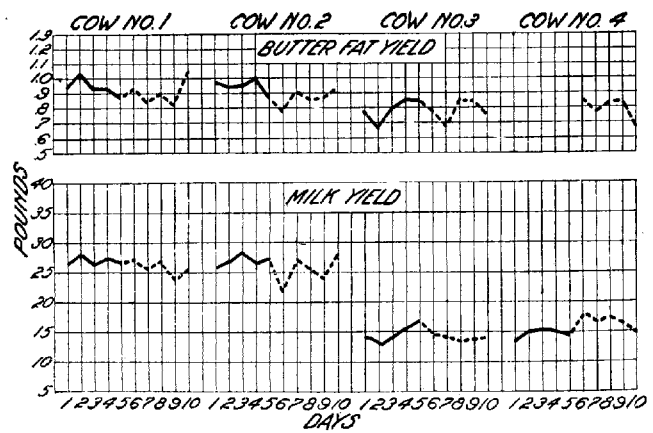

FIG. 4.—Graph showing effect of powdered gentian on butter-fat and milk yield.

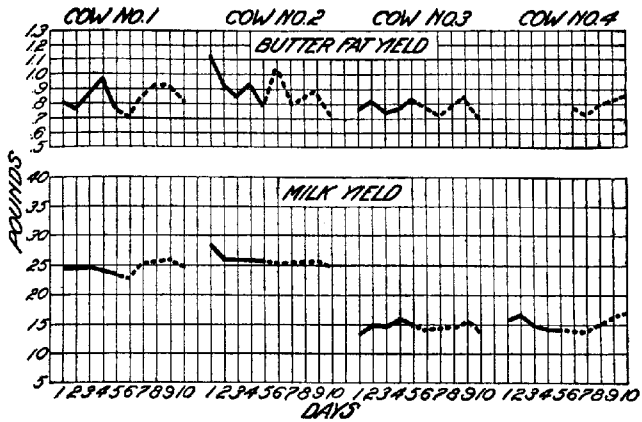

FIG. 5.—Graph showing effect of the German tonic mixture on butter-fat and milk yield.

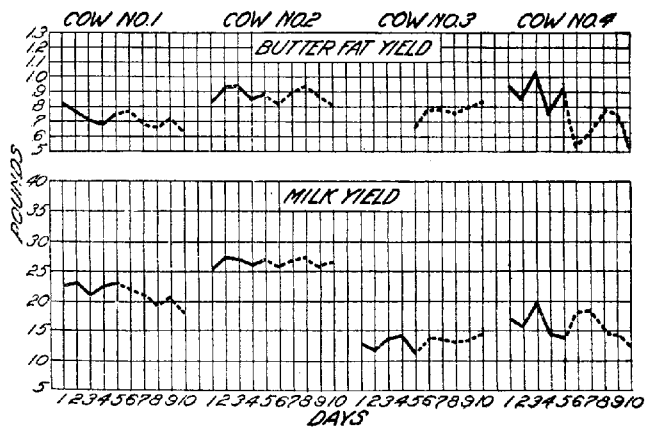

FIG. 6.—Graph showing effect of physostigmine sulphate on butter-fat and milk yield.

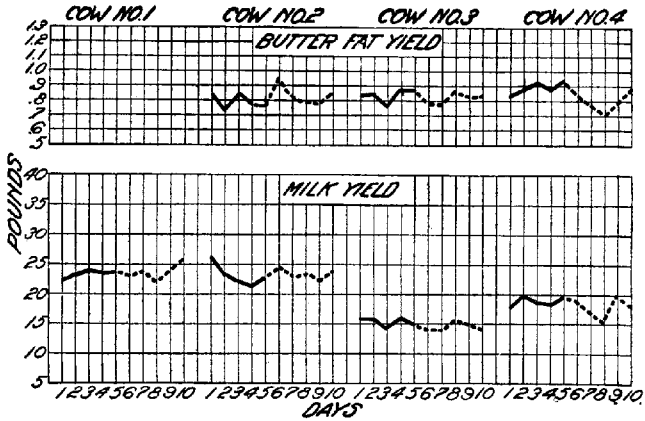

FIG. 7.—Graph showing effect of sodium bicarbonate on butter-fat and milk yield.

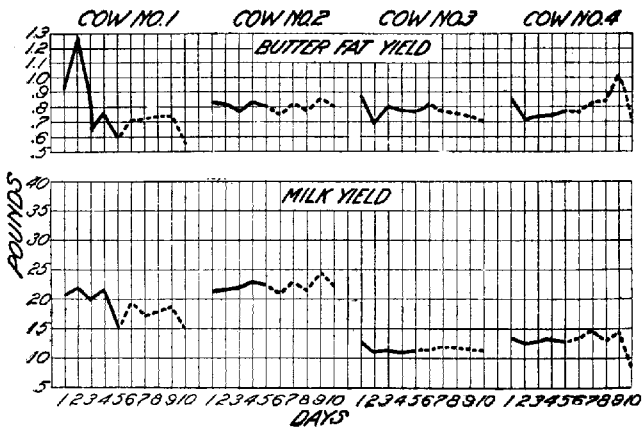

FIG. 8.—Graph showing effect of ginger on butter-fat and milk yield.

ARTIFICIAL AND INSECT TRANSMISSION OF SUGAR-CANE MOSAIC

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The infectious nature of sugar-cane mosaic can hardly be questioned in the light of field observations bearing out this point made in Georgia and Florida last year and in Porto Rico during the preceding two years (*1*) [1]. Records of well-controlled inoculation experiments, however, have been wholly lacking. A number of investigators, beginning with the Dutch workers in Java, have attempted to produce the disease by artificial inoculation and by the use of suspected insect carriers; but in all cases results have been negative or inconclusive. Where success has been reported the experiments were carried on under unsatisfactory conditions, and the results were repudiated by contemporaneous workers who attempted to repeat the experiments. Kamerling (*3*) in 1902 reports that he secured infection by inoculating healthy plants with sap from diseased plants. He says (in translation):

> So far as the kind of disease is concerned, we are dealing with a disease analogous to the notorious mosaic of tobacco, that is, with an infectious disease, which, however, in all probability is not caused by a parasitic organism.
>
> As is the case with tobacco mosaic, the disease has been successfully transmitted by inoculating healthy plants with juice pressed out of diseased plants. (Footnote: My inoculations with juice of diseased cane were performed in the same way as the inoculation tests of Beijerinck with juice of tobacco plants affected with mosaic.)
>
> These inoculation tests, however, throw little light on the manner of origin and of dissemination in nature.
>
> One very great difficulty in carrying out tests on the way in which the disease originates and is disseminated in nature is in securing cuttings that do not have a predisposition toward the disease. From the best possible selected Moga cuttings a certain number of check plants in my pot cultures showed stripe disease; and I have had a similar experience with specially selected cuttings from Van Delden in Soekaboemi, which in Koeningen produced a crop practically free from disease.

This vague reference to his experiments and his admission of disease in the control plants was not very convincing and was discredited by later Dutch investigators. Kobus (*4*), van der Stok (*6*), and Wilbrink and Ledeboer (*7*) were unable to produce the disease by using the method of Kamerling. Wilbrink and Ledeboer say (in translation):

> So sudden a severe outbreak as Kobus already observed gives rise to the suspicion that we are perhaps dealing with an infectious disease, as is the case, for example, with the mosaic disease of tobacco, analogous to the stripe disease in very many respects. Dr. Kamerling states in the Annual Report of the Experiment Station of Kagok for

[1] Reference is made by number (italic) to "Literature cited," p. 138.

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1902 that he succeeded in inoculating healthy plants with the disease by injecting sap from diseased plants. We have repeated these inoculation experiments as far as we have been able to obtain data about them, but without success. Neither have we been able to find any other indication that the disease is contagious.

They conclude with Kobus and van der Stok that the mosaic is an expression of bud variation. No reference is made to successful inoculation experiments in the numerous papers on mosaic in the Hawaiian Sugar Planters' Record for 1911–1919. Stevenson (5) reports hundreds of inoculations of many cane varieties by various methods during 1917 and 1918, all with negative results. Prof. F. S. Earle, in an unpublished paper, calls attention to a method of inoculating with juice expressed under oil to prevent oxidation. Some of the plants he inoculated became diseased, but the experiment was inconclusive and open to the criticism that it was carried on without control plants in a field where cases of the disease were appearing naturally.

Various writers have called attention to the possibility of insect carriers of the mosaic disease, but no published proof has appeared, and the statements have been based on analogy with other apparently similar diseases and on field observations. The failure of all efforts to obtain uniform or dependable results with either artificial methods of inoculation or with insects has been one of the conspicuous peculiarities in the behavior of sugar-cane mosaic. In all inoculation work in plant pathology it is necessary to secure a very high percentage of infection in inoculated plants where control plants are not absolutely protected from extraneous infection. In diseases like cane mosaic, where, for reasons which we are not in a position to discuss at present, the percentage of infection resulting from experimental inoculation is not high, it is not only necessary that all experimental plants be apparently healthy but also that they be of known healthy parentage for at least one generation back and preferably more. Further than this, the experiments should be performed under absolutely controlled conditions. The prevention of contamination of experimental plants with diseased material by direct or indirect contact must be absolute. Special precautions must be taken to prevent the admittance to treated plants of insects or any other animals other than the ones being experimented with.

The writer became convinced, after observations and experiments with the mosaic disease dating from the summer of 1916, that more reliance can be placed on the results of experiments performed in some region far removed from any chance of accidental infection. It was owing to these considerations that the experiments recorded here were performed at a distance from the seat of any natural infection, because the required conditions would be practically impossible to obtain where the disease is prevalent.

The first experiments were conducted in a quarantine greenhouse near Garrett Park, Md. Later experiments were made in several green-

houses at Washington. The insects used were those at hand which were known to feed on sugar cane. Provision has been made by cooperation with the Bureau of Entomology to collect information leading to the identification of the particular insect or insects responsible for secondary infections in the infested cane regions. Mr. George N. Wolcott, of the Bureau of Entomology, is at present working on that phase of the problem in Porto Rico.

EXPERIMENTS AT GARRETT PARK, MD.[1]

Seed pieces from diseased parent stock were received from time to time during 1918 and 1919 and planted in the greenhouse, which was screened with physician's cloth so that insects could not escape. On August 10, 1918, a shipment of diseased Crystalina cane from Ensenada, P. R., was planted. Yellow Bantam sweetcorn and Sugar Drip, Early Amber, and Japanese Ribbon sorghum were planted August 13, 1918, in the same greenhouse. On September 24, 1918, a shipment of diseased Rayada cane from Rio Piedras, P. R., was planted. Diseased seed pieces of Morado, Yellow Caledonia, Crystalina, and Rayada varieties from Arecibo, P. R., were planted on April 24, 1919. Similar pieces of Selangore, D.–117, and Rayada from Mayaguez, P. R., were planted on April 25, 1919. Lastly a shipment from Yauco, P. R., containing diseased seed pieces of G. C.–701, G. C.–1486, B.–3922, B.–6450, and P. R.–260 were planted May 1, 1919.

Through the kindness of Dr. Erwin F. Smith, cuttings of Lahaina cane were secured from plants which had been growing in one of his greenhouses at Washington for more than six years and showed absolutely no signs of mosaic. These cuttings were planted in pots in a third greenhouse at Washington on December 10, 1918. All the cane, diseased and healthy, sprouted and grew well. All cuttings from diseased parents produced mottled sprouts, without exception, and all cuttings from Dr. Smith's healthy cane produced in great contrast healthy plants with leaves of uniform dark green color.

EXPERIMENT 1.—This was a preliminary experiment to determine whether infection could take place by natural means, merely by exposing healthy plants in the same greenhouse with diseased plants. On May 10, 1919, 5 healthy cane plants, 5 months old, in pots were taken from the greenhouse in Washington and placed in the quarantine greenhouse at Garrett Park, Md., in such a way that the leaves did not come in contact with the leaves of diseased plants. At that time the corn aphis (*Aphis maidis*)[2] was abundant on the sorghum. The wild grasses, a few clumps of which came up as weeds in the greenhouse, were infested with red spiders (*Tetranychus binaculatus*). Both these insects were seen occasionally in the cane. A small leafhopper was also seen but was not captured

[1] Thanks are due Dr. Caroline Rumbold, who was in charge of this work during the writer's absence on trips to the Tropics.

[2] Identified by Dr. A. C. Baker, of the Bureau of Entomology, United States Department of Agriculture.

and consequently was not determined. On June 3, 1919, all five of the Lahaina cane plants from Dr. Smith's greenhouse showed unmistakable incipient signs of mosaic. Two weeks later all were well-developed cases.

EXPERIMENT 2.—On July 3, 1919, 15 healthy cane plants of the Lahaina variety, 7 months old, were removed from the greenhouse in Washington to the "pesthouse" at Garrett Park. Five were placed within the house unprotected as before, and 5 were placed in each of two insect-proof cages. On July 22, 4 of the exposed plants showed incipient signs of mosaic. On August 2 the remaining plant showed evidence of being infected, and a week later all the exposed plants exhibited well-advanced leaf symptoms. At this time the 10 control plants in cages were perfectly normal and continued so until they were used in another experiment two months later.

EXPERIMENT 3.—Seeds of sweetcorn (Yellow Bantam variety) and sorghum (Sugar Drip, Early Amber, and Japanese Ribbon) were planted on August 13, 1918, in the Garrett Park quarantine greenhouse. They germinated and grew slowly during the winter, then more rapidly in the spring. A number of volunteer grasses that came up as weeds in the greenhouse were allowed to mature. All these plants soon became heavily infested with corn aphis. Sorghum seed from the same lot was planted in a greenhouse at Washington. On May 7, 1919, a few mottled leaves appeared on the sorghum plants at Garrett Park. Examination of the wild grasses revealed the typical streaking and mottling in practically every stool of crabgrass (*Syntherisma sanguinalis*), foxtail (*Chaetochloa lutescens*) and *Panicum dichotomiflorum*. Other wild grasses in the greenhouse were normal. At this time the sorghum control plants in the Washington greenhouse and the wild grasses of the same species outside the greenhouse at Garrett Park showed no signs of mosaic, nor did they show any evidence of mosaic during the remainder of the summer.

EXPERIMENT 4.—On August 7, 1919, about 50 adult individuals of the sharp-headed grain leafhopper (*Draeculacephala molipes*)[1] collected two days previously on mosaic-diseased sugar cane at Audubon Park, New Orleans, La., were placed in a cage with 5 healthy cane plants at the Garrett Park greenhouse. The leafhoppers immediately began feeding on the healthy cane. No infection was evident after two months.

EXPERIMENTS AT WASHINGTON

During September, 1919, nearly all experiments were transferred to greenhouses especially prepared to receive them at Washington. Ventilators of the 2-story greenhouse, formerly used by Dr. Smith for bananas, were screened with physician's cloth; and the diseased cane plants of all varieties were removed to it from Garrett Park. A greenhouse in another range, separated by a roadway from the first, was screened; and

[1] Identified by Mr. T. E. Holloway, Bureau of Entomology, United States Department of Agriculture.

300 healthy Lahaina cane plants, from cuttings supplied by Dr. Smith, were placed therein. These plants were from the same source as the ones previously mentioned. The second greenhouse was divided into halves by a tight glass partition. One compartment was used for propagating healthy stock, and the other compartment was used for artificial inoculation experiments. Both compartments were kept free from insects by frequent fumigation. In the banana house, or "pesthouse" fumigation was not practiced on account of cage experiments with insects. The greatest precautions were taken to prevent accidental infection of plants in the house where healthy stock was growing. This house was invariably the first one visited by the gardener for routine work such as watering, and both houses were kept padlocked at all times. Probably because of this care no single case of mosaic has appeared there or on control plants in either house in any of the experiments.

INOCULATIONS WITH INSECTS

EXPERIMENT 1.—On October 8, 1919, 10 individuals of *Aphis maidis* were transferred with a camel's-hair brush from mosaic sorghum to each of four young healthy cane plants in separate cages. A fifth cage was reserved for two healthy plants as controls. On October 28 all four plants showed incipient signs of mosaic. On November 18 they were all unmistakable, well-advanced cases. The two control plants remained healthy.

EXPERIMENT 2.—On February 2, 1920, 12 to 15 individuals of *Aphis maidis* were lifted from mosaic sorghum and placed on each of three healthy cane plants in separate cages. Two healthy cane plants were placed in a fourth cage for controls. On February 28 two of the treated plants showed signs of mosaic and on March 5 were typical cases. The two control plants remained healthy.

EXPERIMENT 3.—On February 2, 1920, one mosaic sorghum plant infested with *Aphis maidis* was placed in a cage with a healthy cane plant in such a way that the leaves of the two plants intermingled. On March 21 the cane plant showed unmistakable signs of infection.

EXPERIMENT 4.—February 2, 1920, 10 individuals of *Aphis maidis* were lifted from a diseased cane plant of variety G. C.–701 and placed on a healthy cane plant in a cage. No infection was apparent on March 15.

EXPERIMENT 5.—On October 8, 1919, 15 specimens of *Draeculacephala molipes* were placed in each of five cages containing one healthy and one mosaic cane plants. On January 5, 1920, approximately three months later, there was no evidence of infection.

EXPERIMENT 6.—On January 5, 1920, 15 specimens of *Draeculacephala molipes* were placed in each of two cages containing two mosaic sorghum plants and two healthy cane plants. On March 11 there was no sign of infection on any of the cane plants.

EXPERIMENT 7.—On November 20, 1919, two mosaic cane plants of the Rayada variety, infested with the sugar-cane mealy bug (*Pseudococcus boninensis* (Kuw.),[1] were placed in each of two cages, together with two healthy cane plants of the Lahaina variety. A few of the mealy bugs were transferred from diseased plants to all healthy plants. Ants were assiduously tending the mealy bugs. On March 11, 1920, all healthy plants were badly infested with mealy bugs but there was no mosaic infection.

ARTIFICIAL INOCULATIONS

Virus was obtained for artificial inoculation by two methods. Cell sap from young leaves, designated as virus No. 1, was obtained by grinding the young, tightly rolled leaves of diseased Rayada cane in a food chopper and straining through several thicknesses of cheesecloth. It was used undiluted for inoculating immediately after being prepared. Virus No. 2 consisted of cane juice from the youngest joints, including the growing point. To prevent oxidation this was pressed out under a mineral oil (Nujol) in a specially designed press (*2*). This also was used undiluted as soon as it was prepared. Inoculations were made in the compartment of the fumigated greenhouse separated from all diseased material and protected by every means from accidental infection. The results of these inoculations are given in Tables I and II.

In addition to the control plants injured with a sterile needle, there were about 100 other healthy plants of the Lahaina variety in the compartment. No case of mosaic developed among these plants.

TABLE I.—*Effect of artificial inoculation of Lahaina cane with triturated young leaves (virus No. 1)*

[Plants inoculated Jan. 8, 1920]

Number of plants.	Treatment.	Results.
10	Virus rubbed on unbroken surface of young leaves with fingers.	All healthy Mar. 21.
10	Youngest leaves inoculated by numerous needle pricks.	One mosaic Mar. 21.
5	Control plants pricked with sterile needle	All healthy Mar. 21.
10	Epidermal layer of young leaf cells scarified with sharp needle dipped in virus.	Do.
5	Control plants scarified with sterile needle	Do.
10	Young leaves scarified as above and virus rubbed in vigorously with the fingers.	Do.
10	Inoculated by injecting ½ cc. of virus into growing point with hypodermic syringe.	Two mosaic Feb. 14; eight healthy Mar. 21.
5	Control plants punctured at growing point with sterile needle.	All healthy Mar. 21.

[1] Identified by Mr. Harold Morrison, of the Bureau of Entomology, United States Department of Agriculture.

TABLE II.—*Effect of artificial inoculation of Lahaina cane with juice from cane unoxidized (virus No. 2)*

[Plants inoculated Jan. 7, 1920]

Number of plants.	Treatment.	Results.
10.....	Virus rubbed on unbroken surface of young leaves with fingers.	All healthy Mar. 21.
10.....	Youngest leaves inoculated by numerous needle pricks.	Do.
5.....	Control plants pricked with sterile needle.....	Do.
10.....	Epidermal layer of young leaf cells scarified with sharp needle dipped in virus.	Do.
5.....	Control plants scarified with sterile needle.....	Do.
10.....	Young leaves scarified as above and virus rubbed in vigorously with fingers.	Do.
10.....	Inoculated by injecting ½ cc. of virus into growing point with hypodermic syringe.	Eight mosaic Feb. 6 to 14.
5.....	Control plants punctured at growing point with sterile needle.	All healthy Mar. 21.

DISCUSSION

From the foregoing results it may be inferred that the sugar-cane mosaic virus is highly infectious only when exacting demands in the matter of favorable conditions are satisfied. Erratic spreading under natural conditions in the field also indicates the necessity for special conditions, which are not as yet known. It is considered as proved, however, that the cell sap of diseased plants is infectious when introduced in the proper manner and that the disease can be transmitted by insects. Just what insects are responsible for dissemination in the cane regions remains to be proved. The failure of the sharp-headed grain leafhopper to transmit the disease under the conditions of these experiments is surprising. This insect is very common on cane in Louisiana, and as a result of field observations suspicion was directed toward it from the first. Other leafhoppers are now being tested. The successful experiments with the corn aphis is of great interest scientifically, but it is not believed that transmission of mosaic is restricted to this insect or to other aphids more abundant on cane. *Aphis maidis*, however, has been reported on sugar cane from practically every sugar-cane region in the world.

That cane mosaic is analogous with other mosaic diseases is brought out by a number of facts, aside from the visible signs of the disease. As in many other mosaics, the infectious material does not seem to be highly specialized, but may attack other plants of the same family. The cell sap of infected plants contains some organism, not visible by ordinary means, which is capable of inducing the disease when injected into healthy plants. Leaves which are mature at the time of inoculation never show any signs of mosaic. This fact, typical of all mosaics, has been brought

out in all inoculation experiments with sugar cane. The disease can be transmitted by certain sucking insects. There is no known period of saprogenesis in the existence of the virus. Seed transmission of the virus is one of the phenomena concerning which divergent results have been recorded for the various mosaic diseases. This point has not been definitely settled for sugar-cane mosaic, but mosaic sorghum plants failed to produce mosaic progeny in two experiments.

LITERATURE CITED

(1) BRANDES, E. W.
1919. THE MOSAIC DISEASE OF SUGAR CANE AND OTHER GRASSES. *In* U. S. Dept. Agr. Bul. 829, 26 p., 5 fig., 1 col. pl.

(2) CLARK, W. Blair.
1917. A SAMPLING PRESS. *In* Jour. Indus. and Engin. Chem., v. 9, no. 8, p. 788.

(3) KAMERLING, Z.
1903. DE GELE STREPENZIEKTE DER BLADEREN. *In* Verslag 1902 Proefsta. Suikerriet West Java "Kakok" to Pekalongan, p. 76–81.

(4) KOBUS, J. D.
1908. VERGELIJKENDE PROEVEN OMTRENT GELE-STREPENZIEKTE. *In* Arch. Java-Suikerindus., jaarg. 16, p. 350–374. Reprinted as Meded. Proefsta. Javasuikerindus., no. 12, p. 319–342.

(5) STEVENSON, John A.
1919. THE MOTTLING OR YELLOW STRIPE DISEASE OF SUGAR-CANE. *In* Jour. Dept. Agr. and Labor Porto Rico, v. 4 (i. e. 3), no. 3, p. 3–76, illus., 3 col. pl. Literature cited, p. 73–76.

(6) STOK, J. E. VAN DER.
1907. VERSCHIJNSELEN VAN TUSSCHENRASVARIABILITEIT BIJ HET SUIKERRIET. (PROEVE EENER VERKLARING DER GELE-STREPENZIEKTE EN DER SEREHZIEKTE.) *In* Arch. Java Suikerindus., jaarg. 15, p. 581–601. Reprinted as Meded. Proefsta. Oost-Java, s. 4, no. 36, p. 457–477.

(7) WILBRINK, G., and LEDEBOER, F.
1910. BIJDRAGE TOT DE KENNIS DER GELE STREPENZIEKTE. *In* Arch. Suikerindus. Nederl. Indië, jaarg. 18, p. 465–518. Reprinted as Meded. Proefsta. Java-Suikerindus., deel 2, no. 39, p. 443–495, 5 pl. (4 col.).